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The components of the oxygen debt and the corresponding blood lactate removal during recovery form exercise of varied intensity and duration.

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THE COMPONENTS OF THE OXYGEN DEBT AND THE CORRESPONDING BLOOD LACTATE REMOVAL DURING RECOVERY FROM EXERCISE OF VARIED INTENSITY AND DURATION

BY

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Finally, thanks go to the twelve subjects whose interest in the area, patience and promptness made this study possible.

DEDICATION

This manuscript is dedicted to my loving wife Anita, whose assistance, faith and loving devotion have always been a major factor in any endeavour; to my parents, Charles and Verlie Gardiner, whose encouragement throughout my education has always been unceasing; to my parents-in-law, George and Vilma Turchetto, for their love, understanding and sacrifice through all difficulties, and to my daughter Sandra, who has made all my work worth while.

ABSTRACT

The purpose of the study was to test the Margaria et al. theory of oxygen debt by determining the effect of duration of exercise, at different intensities, on the total oxygen debt and lactic and alactic components of the oxygen debt. In addition, the study proposed to compare the lactic acid removed with the lactic component of the oxygen debt repaid during the same time period, in order to test the stoichiometric relationship inherent in the Margaria et al. theory between these parameters.

Twelve subjects, assigned into three intensity groups, each performed three runs of different durations, and one subject from each intensity group performed two additional special condition runs. Oxygen uptake measures were taken to insure intra- and inter-run consistency for the tests of each intensity group, and continuous oxygen uptake measurements during recovery allowed the calculation of the oxygen debt parameters. Theoretical oxygen debt curves were drawn from mean measurements for each group and duration of running, so that (1) the lactic and alactic components could be derived, and (2) a baseline for the calculation of oxygen debt not involving a possible third component could be utilized. Blood lactic acid concentration was measured at specific intervals during recovery. Analysis of variance and Newman-Keuls Means Tests were used on each dependent variable within each intensity group to test for the effect of duration of exercise, and correlated t-tests were used

to test for differences between the lactic acid removed and the lactic component repaid.

The results rejected the Margaria *et al.* theory on the grounds that; (1) the total oxygen debt did not increase with time above the threshold level proposed by Margaria *et al.* and (2) the lactic component did not increase above the threshold, and did not remain constant below the threshold, as proposed by Margaria *et al.* It also appeared from the results that the stoichiometric relationship between the lactic component and lactic acid removed during recovery proposed was suspect, although, statistically, the theory was upheld.

It was concluded that the oxygen debt phenomenon is more complex than originally conceived, and that assignment of a clearly defined set of biochemical events to a particular is naive at best. In addition, it was concluded that, although the Margaria *et al.* theory was shown in the results of this study to be faulty in many of its tenets, the baseline used for the determination of oxygen debt was not based on a scientifically sound rationale, and therefore the results may be open to question.

Recommendations for further research were proposed which suggest study of the proper baseline for oxygen debt determination, the components of the oxygen debt as they relate to biochemical events during recovery, and the biochemical occurences at the mitochondrial membrane, which, it was hypothesized, hold the key to the mystery of oxygen debt.

TABLE OF CONTENTS

Chapter

I. INTRODUCTION 1

Statement of the Problem
Hypotheses
Alternative Hypotheses
Definition of Terms

II. REVIEW OF THE LITERATURE 12

The Hill-Meyerhof Theory of Oxygen Debt
The Margaria Theory of Oxygen Debt
Opposition to the Margaria Theory
Conclusions from the Literature

III. METHODOLOGY 34

Testing Procedures
Preliminary Testing Procedures
Submaximal Testing Procedures
Measurement Techniques
Statistical Procedures

IV. RESULTS 45

Exercise Oxygen Uptake
Validation of the Theoretical Oxygen Debt Curves
Actual Total Oxygen Debt
Actual Total Oxygen Debt from Minute 6 to Minute 29
Lactic Component (6-29) vs. Lactic Acid Removal
The Alactic Component
Special Conditions

V. DISCUSSION 88

The Baseline for Oxygen Debt Determination
Exercise Oxygen Uptake
Total Oxygen Debt
Lactic Component
Alactic Component
The Relationship of Lactic Acid Removal Versus Oxygen Debt
Special Conditions
The Hypotheses

VI. SUMMARY AND CONCLUSIONS

General Conclusions
Recommendations for Further Research

APPENDIX A 107

APPENDIX B 111

BIBLIOGRAPHY 120

LIST OF TABLES

Table

1. Characteristics of Subjects and Assignment into Groups 46

2. Analysis of Variance of Subject Age 47

3. Analysis of Variance of Subject Maximum Oxygen Uptake 47

4. Analysis of Variance of Subject Weight 48

5. Mean Exercise Oxygen Uptake Measurements 48

6. Analysis of Variance of Mean Percentages of Maximum Oxygen Uptake 50

7. Newman-Keuls Means Test of Percentages of Maximum Oxygen Uptake 50

8. Analysis of Variance of Group A Exercise Oxygen Uptake Data: Minute Four 51

9. Newman-Keuls Means Test of Group A Exercise Oxygen Uptake Data: Minute Four 51

10. Analysis of Variance of Group A Exercise Oxygen Uptake Data: Minute Nine 52

11. Analysis of Variance of Group A Exercise Oxygen Uptake Data: Minute Fourteen 52

12. Analysis of Variance of Group B Exercise Oxygen Uptake Data: Minute Four 53

13. Analysis of Variance of Group B Exercise Oxygen Uptake Data: Minute Nine 53

14. Analysis of Variance of Group B Exercise Oxygen Uptake Data: Minute Fourteen 54

15. Analysis of Variance of Group C Exercise Oxygen Uptake Data: Minute Four 54

16. Analysis of Variance of Group C Exercise Oxygen Uptake Data: Minute Nine 55

17. Analysis of Variance of Group C Exercise Oxygen Uptake Data: Minute Fourteen 55

18. Analysis of Variance of Group A Exercise Oxygen Uptake Data: Fifteen Minute Run 56

19. Analysis of Variance of Group A Exercise Oxygen Uptake Data: Thirty Minute Run 56

20. Analysis of Variance of Group B Exercise Oxygen Uptake Data: Fifteen Minute Run 57

21. Analysis of Variance of Group B Exercise Oxygen Uptake Data: Thirty Minute Run 57

22. Analysis of Variance of Group C Exercise Oxygen Uptake Data: Fifteen Minute Run 58

23. Analysis of Variance of Group C Exercise Oxygen Uptake Data: Thirty Minute Run 58

24. Analysis of Variance of Group A Last Minute Oxygen Uptake 59

25. Analysis of Variance of Group B Last Minute Oxygen Uptake 59

26. Analysis of Variance of Group C Last Minute Oxygen Uptake 60

27. Comparison of Actual Total Oxygen Debt Means Versus Theoretical Oxygen Debt Means For Each Group and Duration 62

28. Correlated t-Tests of Actual Total Oxygen Debt Versus Corresponding Theoretical Oxygen Debt . 62

29. Individual Actual Oxygen Debt From Minute 6 to Minute 29 of Recovery 64

30. Theoretical Oxygen Debt From Minute 6 to Minute 29 of Recovery 65

31. Correlated t-Tests of Actual Oxygen Debt From Minute 6 to Minute 29 Versus the Corresponding Theoretical Oxygen Debt 65

32. Actual Total Oxygen Debt 67

33. Analysis of Variance of Actual Total Oxygen Debt After Three Durations of Exercise: Group A . . 68

34. Analysis of Variance of Actual Total Oxygen Debt After Three Durations of Exercise: Group B . . . 68

35. Analysis of Variance of Actual Total Oxygen Debt After Three Durations of Exercise: Group C . . . 69

36. Analysis of Variance of Actual Oxygen Debt From Minute 6 to Minute 29 of Recovery: Group A . . . 69

37. Analysis of Variance of Actual Oxygen Debt From Minute 6 to Minute 29 of Recovery: Group B . . . 72

38. Newman-Keuls Means Test of Actual Oxygen Debt Means From Minute 6 to Minute 29 of Recovery: Group B . 72

39. Analysis of Variance of Actual Oxygen Debt From Minute 6 to Minute 29 of Recovery: Group C . . . 73

40. Lactic Portion of the Oxygen Debt (Min 6 to 29) Versus Oxygen Equivalents of Lactic Acid Removed 75

41. Correlated t-Tests of the Oxygen Equivalents of the Lactic Acid Removed During Minute 6 to Minute 29 of Recovery Versus the Corresponding Actual Oxygen Debt 76

42. Total Oxygen Debt and the Estimated Alactic and Lactic Components Obtained From the Theoretical Curves 80

43. Actual Total Oxygen Debt, Actual Oxygen Debt From Minute 6 to Minute 19, and O_2 Equivalents of Lactic Acid Removed Following Special Conditions 82

44. Individual Lactic Acid Data: Group A 107

45. Individual Lactic Acid Data: Group B 108

46. Individual Lactic Acid Data: Group C 109

47. Lactic Acid Data for Special Conditions 110

LIST OF ILLUSTRATIONS

Figure

1. Lactate Concentration at End of Exercise vs. Percentage of Maximum VO_2 19

2. Relationship of Lactate, NAD, and Oxygen 31

3. Sample Protocol for Special Condition Run. . . . 38

4. Example of Theoretical Curve 63

5. Actual Oxygen Debt (min. 6-29) vs. Duration of Exercise 71

6. Absolute Underestimation of Oxygen Debt (6 to 29) by Lactic Acid O_2 Equivalents vs. Duration of Exercise 77

7. Relative Underestimation of Oxygen Debt (6 to 29) by Lactic Acid O_2 Equivalents vs. Duration of Exercise 78

8. Estimated Alactic Component vs. Duration of Exercise 81

9. Actual Total Oxygen Debt vs. Special Condition . 84

10. Actual Oxygen Debt (6 to 19) vs. Special Condition 85

11. O_2 Equivalents of Lactic Acid Removed (6 to 19) vs. Special Condition 86

12. Deviation of O_2 Equivalents of Lactic Acid Removed vs. Special Condition 87

CHAPTER I

INTRODUCTION

Two concepts exist that express conflicting opinions as to the exact mechanisms of oxygen debt. The concepts are irreconcilable, and involve the relationships that appear to exist between the oxygen debt after exercise of varied intensity and duration, and the corresponding lactic acid disappearance during the same recovery period. Examination of the two concepts clearly accentuates the need for research which can either support or refute either one, or both of the theories.

In 1907, Fletcher and Hopkins (18) reported from their experiments on isolated muscle preparations that lactic acid was produced during fatigue. Their results demonstrated four fundamental points:

(1) muscle was able to contract normally in the absence of oxygen,

(2) lactic acid was produced during anaerobic conditions, and it accumulated with continued stimulation until the muscle became fatigued and could no longer contract,

(3) when fatigued muscle was placed in oxygen, lactic acid disappeared and the muscle became functional again,

(4) less lactic acid was formed in muscle contracting

in oxygen than in muscle contracting under anaerobic conditions (18).

Since 1907, primarily due to scientists such as Embden and Meyerhof (12) the glycogen-to-lactic acid pathway of anaerobic muscle metabolism has been substantially clarified.

The first published data dealing with the excess oxygen consumption following exercise made no attempt at inferring any possible relationship between this phenomenon of exercise consumption and the results published by Fletcher and Hopkins. In 1920, Krogh and Lindhard (43), observed marked change in the rate of oxygen consumption during this recovery period and attributed it to the oxygen deficit that they had observed during the transition from rest to work (43).

A. V. Hill (30) in 1924, coined the term "oxygen debt" for the phenomenon of paying back oxygen that had been unavailable during exercise, and had been "borrowed" from other energy sources. Hill's research marked the beginning of oxygen debt theory. Unlike the research of Krogh and Lindhard, who merely described the recovery curve following exercise, Hill proposed a relationship between the excess oxygen consumption following exercise and the anaerobic biochemical events that had been described by Fletcher and Hopkins from their isolated muscle data. Hill's theory of oxygen debt attributed the excess oxygen consumption during recovery to the delayed oxidation of lactic acid.

In addition to being the first researcher to boldly infer this relationship, Hill also attempted to describe the shape of the recovery curve in terms of lactic acid oxidation. The fact that the oxygen consumption following exercise did not follow a linear decrease was not a new discovery; Krogh and Lindhard (43) had made note of that phenomenon years before. However, Hill postulated that the "rapid" phase of the oxygen debt curve was due to intra-muscular glycogen formation, while the "slow" phase involved the oxidative removal of lactic acid that had diffused into the plasma and was resynthesized elsewhere, presumably in the liver (30).

Following Hill's work, the first concerted attempt to understand and develop a sound theory of oxygen debt was undertaken at the Harvard Fatigue Laboratory in the 1930's. Margaria and co-workers (49) concurred with the Hill idea of a two-phase oxygen debt curve, but modified the concept by postulating different roles for each component; according to Margaria, the "slow" component was due to lactate removal while the "fast" component was "alactic" in nature and involved other unknown processes and metabolites. Margaria et al. (49) also added that each component appeared to be an exponential function; the fast portion having an apparent half-time of about thirty seconds and the slow component, a half-time of fifteen minutes. Furthermore, their particular theory of oxygen debt stressed that a critical level of exercise existed below which no

lactic acid production, and therefore, no lactic debt occurred during recovery[1].

Although these two theories of oxygen debt and the relationship of recovery oxygen and lactic acid removal have been received with mixed emotions by physiologists, there appears to be a general agreement on the Margaria et al. theory as opposed to Hill's theory of oxygen debt. The idea of a portion of the oxygen debt not related to lactic acid removal has been fairly solidly substantiated using human subjects (19,25,26,53), animals (9) and animal muscle preparations (14,54,62). It has been shown that oxygen during the recovery period can be used in such alactic mechanisms as creatine phosphate resynthesis (3,14,54), body temperature and adrenalin mechanisms (42), refilling of oxygen stores, such as tissue oxygen, oxygen dissolved in the blood, and myoglobin (3,53), the work of breathing and circulatory stress (70), and ion redistribution (42, 61). Similarly, research by Henry (25) and Henry and Demoor (26,27), in their mathematical description of the recovery curve, is ample proof of the two-component exponential nature of the oxygen debt curve.

[1]Margaria et al. (49), in the original paper, concluded that no lactic debt and no lactic acid production occurred below the threshold level of approximately two-thirds of maximum oxygen uptake. Had he studied the lower level oxygen debt curves in more depth, he probably would have found that the lactic component, however insignificant still existed to some degree. Since the work of Margaria et al., Henry and DeMoor have shown that a small lactic component exists following exercise requiring less than one liter of oxygen per minute.

More recently, a third theory of oxygen debt has evolved as a result of research by Knuttgen (41,42), Kayne and Alpert (39), Alpert, Kayne and Haslett (2) and Rowell (56). The results of the data from these studies point invariable to the same conclusion; that there appears to be no causal relationship between oxygen debt and lactic acid removal, and that any apparent relationship is merely coincidental. Although no alternative hypothesis to Margaria et al.'s, or Hill's theories have been proposed, such factors as body temperature, the versatility of lactic acid as substrate in resting tissues, and substrate diffusion difficulty at the mitochondrial level, have all been suggested as possible reasons for the lack of a stoichiometric relationship between oxygen debt and lactic acid removal.

It appears, therefore, that three theories of oxygen debt exist at the present time. Although Hill's oxygen debt theory has been shown to be unsound (25,39,42,56), there has been a lack of research conducted to determine which of the two alternative theories is acceptable. In particular, the study of the relationship between the lactic component of the oxygen debt and the disappearance of lactic acid during the same time period, has been neglected.

As pointed out previously, the theory of Margaria et al. (49) upholds the concept that there is a critical level of exercise, approximately two-thirds of the maximum oxygen consumption, below which there is neither lactic acid production, nor lactic component of the oxygen debt. Above this threshold level, according to the theory, lactic acid

is produced during exercise, and a corresponding lactic debt is incurred. As maximum oxygen uptake is approached, the rate of lactic acid production increases. It logically follows that, below the critical level of exercise, the size of oxygen debt is independent of the duration of exercise. Similarly, above the critical level, oxygen debt increases as the duration of the exercise increases, since lactic acid is produced at a constant rate during the exercise. The lactic acid response, according to the theory, follows the same trends.

However, data have been published which indicates that this may not be the case, Saiki (57) showed that lactic acid levels rose and then returned to pre-exercise values in subjects exercising at seventy to eighty percent of maximum aerobic capacity, while Wasserman (68) and DiPrampero (15) found lactic acid increased during exercise of less than forty percent of maximum. Similarly, the fact that long distance skiers (4) and runners (11) have been shown to have very low blood lactate levels at the end of competition despite physical exhaustion warranted the study of the effect of duration of exercise on lactic acid accumulation and the corresponding lactic portion of the oxygen debt at different intensities of exercise.

Statement of the Problem

The purpose of this study was to test the Margaria *et al.* theory of oxygen debt by determining how oxygen debt and lactic acid removal are affected by duration of exercise

at various submaximal intensities.

More specifically, the study attempted to investigate the following subproblems:

(1) The effect of duration of exercise at different intensities on the total oxygen debt, and on the estimated lactic and alactic portions of the oxygen debt.

(2) The comparison of the lactic acid removed from the blood during recovery with the lactic component of the oxygen debt repaid during the same time interval.

(3) The comparison of the total oxygen debt, and the lactic component of the oxygen debt, as affected by the time a short period of supramaximal work was added during "steady state" exercise.

Problems 1 and 3 are based on a concept inherent in the theory of Margaria et al. (49) and expressed more recently by Schneider et al. (59), that, under a specific threshold level of exercise (two-thirds of maximum aerobic capacity) the oxygen uptake level during that exercise is the result of the satisfaction of the oxygen need at the tissue level. Once this need is satisfied, the only debt that is repaid following exercise is that debt resulting from anaerobic metabolism that occurred during the first minutes of exercise. In conflict with this concept, is the one expressed by Stainsby and Barclay (61) and Rowell (56), that the oxygen consumption during exercise of a relatively mild to moderate intensity is a reflection of both tissue need and metabolic turnover, which implies the repayment of

oxygen debt during mild exercise.

Hypotheses

For the purposes of investigating the problem, the following set of hypotheses was constructed:

(1) It was hypothesized that variations in the length of time at a given submaximal intensity of exercise, below the two-thirds of maximum oxygen uptake threshold specified by Margaria, would result in no significant change in the subsequent total oxygen debt (H_1: $a_1 = a_2 = a_3$).

(2) It was hypothesized that variations in the intensity of exercise below this threshold would result in no significant differences in the subsequent estimated lactic component of the oxygen debt (H_2: $b_1 = b_2 = b_3$).

(3) It was hypothesized that, above the two-thirds of maximum threshold, the total oxygen debt would increase as a result of increasing the duration of exercise (H_3: $C_1 < C_2 < C_3$).

(4) It was hypothesized that, above the two-thirds of maximum threshold, the lactic component of the oxygen debt would increase as a result of increasing the duration of exercise (H_4: $d_1 < d_2 < d_3$).

(5) It was hypothesized that, irrespective of intensity and duration, the lactic acid removed during a specific interval during recovery, expressed as oxygen equivalent values, would exist in a stoichiometric relationship with the lactic component of the oxygen debt repaid during the same time interval.(H_5: $e_1 = e_2$).

In addition to these five hypotheses, the following subproblems were investigated, with no statistical analysis, in order to aid in the clarification of the relationships to be tested statistically:

(6) To evaluate the effect of duration of exercise, at each intensity of exercise studied, on the estimated alactic component of the oxygen debt.

(7) To evaluate the effect of adding a short, maximal bout of exercise at two different periods during a steady state run on the total oxygen debt and the lactic component of the oxygen debt.

(8) To evaluate the effect of this change in the time of presentation of the maximal bout of running on the relationship between the lactic component and the removal of lactic acid during the same period of time.

Alternative Hypotheses

In the event of the rejection of any or all of the hypotheses proposed above, the following set of alternative hypotheses was constructed:

1. H_0: $a_1 \neq a_2 \neq a_3$
2. H_0: $b_1 \neq b_2 \neq b_3$
3. H_0: $C_1 = C_2 = C_3$
4. H_0: $d_1 = d_2 = d_3$
5. H_0: $e_1 \neq e_2$

Definition of Terms

Oxygen Debt

In spite of the various baselines that are frequently used to determine oxygen debt, the term generally refers to "the oxygen uptake of the subject during recovery, in excess of the oxygen uptake calculated for an equivalent period of rest not preceded by activity" (3). However, in this study, oxygen debt was redefined as the excess oxygen consumption during the recovery period immediately following exercise minus the asymptote calculated from the recovery uptake values when plotted on linear graph paper. This redefinition of oxygen debt made the study unique in that the two-exponential nature of the oxygen debt curve was accentuated, allowing more detailed examination of the lactic component with respect to the blood lactic acid disappearance. In addition, a possible third component of the oxygen debt, involving the restoration of normal body temperature, ion redistribution, and other prolonged consequences of exercise, was prevented from interfering with the two components examined, which has been a considerable setback in previous research attempts.

Lactic Acid Production and Removal

The production of lactic acid from glucose is the overall result of glycolysis in skeletal muscle, with the generation of a small amount of usable energy in the form of ATP:

$$\text{Glucose} + 2\text{NAD} \longrightarrow 2 \text{ Pyruvic acid} + 2\text{ATP} +$$

$$2NADH_2 \longrightarrow 2 \text{ Lactic acid} + 2NAD$$

In the absence of adequate oxygenation, oxidation of the NAD necessary for the continuation of the glycolytic process is provided by the reduction of pyruvate to lactate. During recovery, when adequate oxygen is once again made available to the tissues, lactic acid is oxidized by NAD to form pyruvic acid and $NADH_2$, which is, in turn, oxidized by molecular oxygen (21).

Actual Oxygen Debt

This is a phrase coined for purposes of this study, and refers to the excess oxygen consumption during recovery from exercise, as obtained from summing the individual recovery interval oxygen consumption data, minus the asymptote.

Theoretical Oxygen Debt

This term refers to the oxygen debt as determined from the theoretical curves, using Henry's formula (25):

$$Q = {}^{a}1/k_1 + {}^{a}2/k_2 + C$$

where Q equals the total oxygen debt, a_1 and a_2 refer to the amounts of the alactic and lactic component, respectively, at time zero of recovery, k_1 and k_2 refer to the alactic and lactic recovery velocity constants, and C designates the determined asymptote.

CHAPTER II

REVIEW OF THE LITERATURE

The present study was conducted for the purpose of investigating the oxygen debt theory of Margaria et al. (49) with reference to: (1) the two-thirds of maximum oxygen uptake threshold level which is said to be the point at which oxygen debt accumulates over time; and (2) the stoichiometric relationship which is implied between lactic acid removal during recovery and the lactic component of the oxygen debt.

Review of the literature reveals a major dichotomy of thought with respect to oxygen debt theory. With the exception of a few scientists who claim that their data uphold the original Hill theory of oxygen debt, the major split is between the theory as originally postulated by Margaria et al. and the concepts proposed by more recent investigators which are in direct conflict with the Margaria theory.

The Hill-Meyerhof Theory of Oxygen Debt

From his exploratory research in the 1920's related to the oxygen debt phenomenon and lactic acid removal during recovery from exercise, A. V. Hill (30) attributed a major portion of the excess oxygen consumption in

recovery to the formation of glycogen. According to the Hill-Meyerhof theory of oxygen debt, three-quarters of the lactic acid removed was resynthesized to glycogen and one quarter was oxidized to carbon dioxide and water. The differences in the rate of the disappearance of the lactate and excess metabolic rate were considered to be the result of two phenomena:

(1) the "rapid" phase, due to intra-muscular oxidation of lactic acid,

(2) the "slow" phase, involving the removal of lactate that had diffused into the plasma and was oxidized elsewhere in the body (30).

Hill and co-workers were well aware of the two component nature of the oxygen debt; however, they were stubborn in their belief that there was only one mechanism to explain the excess oxygen consumption during recovery; that mechanism according to Hill et al., was the oxidation of lactic acid formed during the exercise.

Although the Hill-Meyerhof theory has been shown, since its conception, to be faulty in many respects, there has been data published which tends to uphold the basic concepts of this theory. Huckabee (31) in 1958, proceeded to oppose the theories of oxygen debt that were currently accepted by supporting Hill's theory that the entire oxygen debt was due to the removal of lactic acid. After finding that lactic acid production in humans could be stimulated by such non-hypoxic situations as hyperventilation, and

the infusion of pyruvate and sodium bicarbonate, Huckabee introduced his formula for "excess lactate", based on the ratio of lactate to pyruvate at rest and the kinetics of the lactic dehydrogenase system, with which he claimed to be able to find the lactate produced solely as a result of exercise. Using this formula, Huckabee found that "excess lactate" was produced at all intensities of exercise, and found significant relationships between calculated "excess lactate" oxygen equivalents and the excess oxygen uptake during recovery. Although more complex in its concepts and methodology than Hill's research, the work of Huckabee concurred with Hill's data from thirty-six years before; that recovery oxygen was being utilized for the removal of lactic acid.

However, data have been published showing, rather conclusively, that the theories of oxygen debt proposed here may be faulty. For example, work conducted by Wasserman (69), Knuttgen (41) and Thomas et al. (66), which tested Huckabee's and consequently Hill's concept of oxygen debt, found that the measured recovery oxygen always surpassed the amount of oxygen equivalent of the excess lactate. They concurred in their conclusions that there was a portion of the oxygen debt not associated with the removal of lactic acid. This concept is one of the basic concepts of the Margaria theory of oxygen debt.

The Margaria Theory of Oxygen Debt

In 1934, Margaria, Edwards and Dill (49) observed that exercise (treadmill running) could be carried out at low levels of intensity without significant changes in resting levels of blood lactate. They found lactate increase above a running speed that elicited an oxygen consumption above two-thirds of maximum aerobic capacity, with a direct and curvilinear relationship existing between lactic acid concentration and magnitude of the oxygen debt.

It was on the basis of these results that Margaria and co-workers concluded that the oxygen debt consisted of two distinct components involving different metabolic processes; the slow component was due to lactate removal while the fast component was alactic in nature and involved other unknown processes and metabolites. They also added that each component of the oxygen debt curve appeared to be an exponential function; the fast and slow components having apparent half times of about thirty seconds and fifteen minutes, respectively.

From the results of later follow-up studies (46,47,48, 50) Margaria and co-workers added to their original concepts of oxygen debt. In 1963, Margaria et al. (48) intensively examined the kinetics and mechanisms of oxygen debt contraction and lactic acid production as it related to time and intensity of exercise. They substantiated their previous claims regarding the alactic versus lactic components of the debt, and also found that, above the

two-thirds of maximum level of exercise, lactic acid concentration, and therefore, oxygen debt values, were higher after longer runs at the same intensity.

In summing up the basic concepts of the Margaria theory of oxygen debt, the following points stand out:

(1) Oxygen debt is comprised of an alactic and lactic component.

(2) No lactic acid production occurs when the exercise is below the two-thirds of maximum threshold level;

(3) If the duration of the exercise period above the two-thirds of maximum threshold level is increased, the lactic acid produced by that exercise, and consequently the oxygen debt also increase.

It is not difficult to comprehend how such a theory would be in direct confrontation with the theory proposed by Hill and Meyerhof.

More recent research has been carried out which tends to support Margaria's contention of a two-component oxygen debt. In 1951, Henry (25), and later Henry and DeMoor (26, 27), on the basis of biochemical considerations, formulated an exponential equation that could be used to describe the total oxygen debt curve in terms of the alactic and lactic components:

$$y = a_1 e^{-k_1 t} + a_2 e^{-k_2 t}$$

where: y = rate of net oxygen intake during recovery

at any time t

a_1 = point on the y axis where alactic exponential line intercepts

a_2 = point on the y axis where lactic exponential line intercepts

$a_1 e^{-k_1 t}$ = alactic component

$a_2 e^{-k_2 t}$ = lactic component

In their studies of the recovery curves following exercise requiring from .21 to 2.1 litres of oxygen per minute, they found that their data fit the mathematical formulae which they had proposed. Furthermore, they introduced formulae for determining the amount of alactic and lactic component knowing the y-axis intercepts and the respective velocity constants of each component.

Knuttgen (41), in his research on excess lactate and oxygen debt, supported the earlier concept of both an alactic and a lactic oxygen debt. Similarly, Barnard, Foss and Tipton (7), after conducting a study in which they poisoned the enzyme systems involved in lactic acid removal in animals, were forced to conclude that the debt consisted of more than merely the removal of lactic acid.

With respect to Margaria's ideas concerning the threshold for the production of lactate, a number of published research articles have shown the threshold to exist. For example, Schneider et al. (59) in a study involving rates of work from thirty to fifty-five percent of maximum

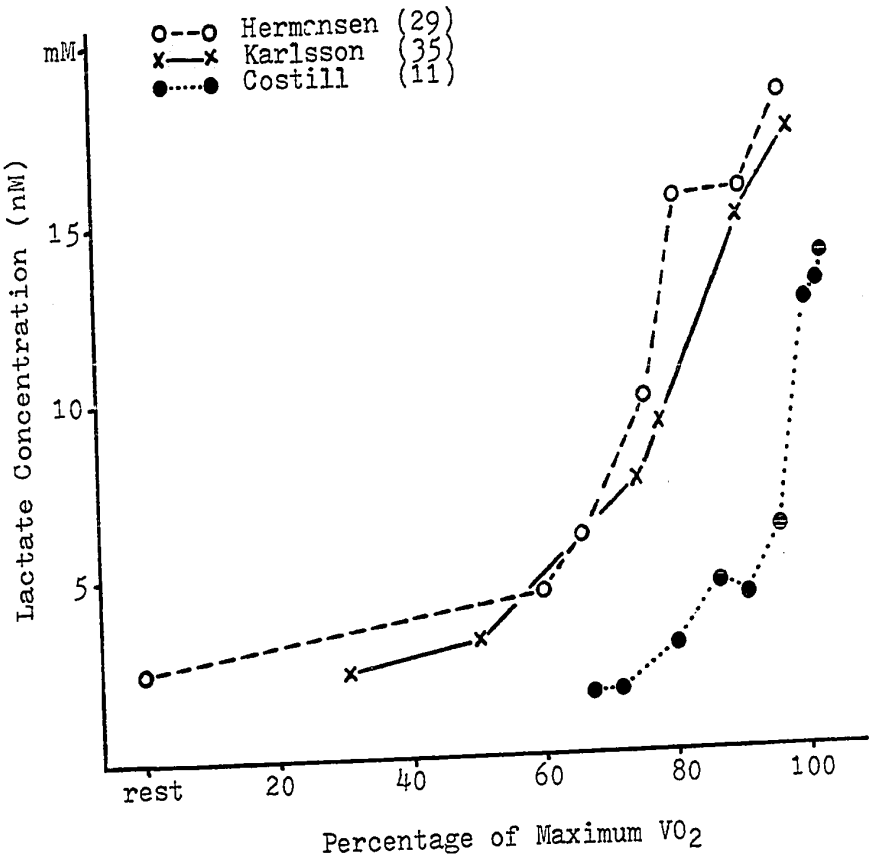
aerobic capacity, found no significant rise in blood lactic acid and, correspondly, no increase in oxygen debt as the exercise was continued from three to twenty-five minutes. Similar data reported by Hermansen (29), Karlson (35), and Costill (11) are presented in Figure 1.

Measurements taken during intermittent exercise have also been instrumental in promoting the acceptance of the alactic concept. Margaria (50), by adjusting the rest period between supramaximal bouts of work of ten seconds duration, found that the half-time of the alactic mechanism was twenty to twenty-five seconds, which was in support of previous estimates. He also concluded that the alactic mechanism chronologically preceded the lactic debt during the recovery period, and that the lactic debt repayment did not significantly come into play until the alactic debt was repaid, or reached a critical level of repayment. Fox et al. (19) in their studies on intermittent exercise, found that for any given level of energy expenditure, a higher portion of the energy was provided by the alactic and a lower portion by the lactic during the intermittent exercise.

In spite of the research previously reviewed which supports Margaria's contention that the oxygen debt is a two-component exponential function, it has been primarly due to alactic metabolite research in the past few years that the two-component relationship has been finally accepted widely.

Hultman, Bergstrom and Anderson (32) in 1967 were

Figure 1:--Lactate Concentration at End of Exercise vs. Percentage of Maximum VO_2



among the first to investigate the utilization of PC and ATP during muscular work in man. In their initial investigations they examined these parameters before, during and after bicycle ergometer exercise of varied intensities and durations. From their data they concluded that a considerable oxygen debt without lactate production occurred due to the breakdown of active phosphate in working muscle. Similarly, Pearl et al. (53), in their study on dogs, found a relationship between oxygen deficit and the depletion of high energy phosphate (increase in phosphate acceptor). Piiper and Spiller (54) conducting research with isolated dog gastrocnemius muscle, attempted to determine (a) if the time course of regeneration of high-energy phosphates correspond to that of oxygen debt repayment, and (b) if the size of the oxygen debt repaid was energetically sufficient to explain the resynthesis of high energy phosphates. Their data illustrated that the major part of the oxygen debt in the isolated muscle group was repaid in two minutes (seventy-seven percent of the total, and ninety-nine percent of the fast component). Since the resynthesis of high energy phosphates was practically completed in two minutes, they postulated that the increase in high energy phosphate concentration and the oxygen debt repaid during the same time interval were related. Thus, the translation of Margaria's alactic concept into metabolite data was a major factor in the acceptance of the two-component theory of oxygen debt.

Other factors that have been shown to be involved in the alactic mechanism of oxygen debt include body temperature (42), adrenalin secretion during and following exercise (42), the refilling of oxygen stores, such as tissue oxygen, oxygen dissolved in the blood, and myoglobin (3,53), the work of breathing and circulatory stress (70), and ion redistribution (42,61). Although the interaction of all of these factors during and after exercise and their influence on recovery oxygen has not been delimited, their existence provides ample proof that mechanisms other than lactic acid removal are significant in oxygen debt.

In summing up the theory of Margaria et al. regarding oxygen debt and the implications that it holds for this study, the following concepts are evident:

(1) There is no increase or decrease in blood lactic acid or oxygen debt over time when the exercise performed is under the threshold levels proposed earlier. This idea was expressed by Schneider et al. (59), who proposed that the level of oxygen consumption during sub-threshold exercise was dependent on the maintenance of a constant oxygen debt as the work was continued, and that the oxygen consumption at any given time during exercise was a measure of the oxygen need at the tissue level, irrespective of the oxygen debt incurred during the initial stages of exercise.

(2) Above the threshold level of exercise, there is a gradual increase in lactic acid levels in the blood, and

a corresponding increase in oxygen debt, to remove this lactic acid, as the duration of the steady state exercise increases.

However, research aimed at testing these concepts has shown diverse results. The investigators publishing results that have compared favourable with the Margaria theory have been presented. The next section outlines the research that has been published which opposes the Margaria theory, and which tends to weaken many of the pre-conceived concepts of oxygen debt mechanisms.

Opposition to the Margaria Theory

The literature which directly opposes the fundamental concepts of the Margaria theory can be grouped into three major areas: (1) that research dealing with the production and removal of lactic acid during and following exercise; (2) that research which has studied the repayment, maintenance or accumulation of oxygen debt following submaximal exercise; and (3) that research which has attempted to show that the relationship between oxygen debt and lactic acid removal, originally proposed by Margaria *et al.* is a tenuous one.

1. Lactic acid production and removal

In opposition to the Margaria threshold concept of lactic acid production, investigators have reported data indicating that lactic acid production can occur at levels of exercise below this threshold. DiPrampero (14), for

example, found, in his isolated muscle preparations, that there was lactic acid production even at twenty to twenty-five percent of maximum aerobic capacity; lactate concentration climbed to a plateau above pre-exercise and remained there until the end of the exercise period (seven minutes). DiPrampero explained this by hypothesizing that this production was due to the impaired oxygen transport resulting from the surgical technique involved in his research (14).

Similarly, research has brought forth results indicating that, above threshold, lactic acid production may not increase as described by Margaria et al. Rowell et al. (56), Saiki et al. (57) and Costill (11) reported a gradual removal of blood lactate over prolonged periods of submaximal exercise above two thirds of maximum aerobic capacity. This disappearance of the blood lactate was attributed to either repayment of part of the lactic acid portion of the oxygen debt during exercise, or oxidative removal of the lactate by resting muscles and other organs: these data were not supported by accompanying oxygen debt data. However, the important concept put forth was that, with exercise above the two thirds of maximum threshold, lactate production was dependent upon the length of time during which exercise was performed, and the longer the duration of exercise, the closer the lactic acid level was, at the end of exercise, to the pre-exercise values.

Studies involving lactic acid measurements following exhaustive exercise have found comparable results.

Karlsson et al. (38) in an experiment in which subjects were exercised until exhaustion for three, seven and twenty minutes, reported that the lactic acid levels at the end of the exercise bouts were lower, the longer the exhaustive exercise periods were performed. From their results, Karlsson et al. concluded that if local lactate in the muscle was the limiting factor for physical performance at the two highest work loads (exhaustion in three and seven minutes) some additional factor must be present at the lowest load sensitizing the muscle to the lactate concentration; for example, a decreased buffering capacity of muscle due to prolonged exposure to metabolic acids. According to their data, mechanisms other than lactic acid production and removal must be involved in "lactic" debt accumulation and repayment. Similar results have been reported by Hermansen (29).

Saiki (57) in 1967, studied the blood lactic acid production in men performing exercise at seventy to eighty percent and ninety to one hundred percent of maximum oxygen uptake. In men performing at the lower level, the blood lactate increased in the first two to five minutes, then tended to pre-exercise levels, while during the higher exercise intensity, lactate increased to a higher value and levelled off. He concluded from his results that there was no appreciable lactate production during submaximal work once steady state oxygen uptake was reached, thus refuting the threshold concept. In addition, Saiki

hypothesized that, since lactate disappeared during the seventy to eighty percent exercise period, the oxygen uptake at equilibrium was sufficient, not only to supply the energy for actual work performed, but also to provide for the energy necessary to reconvert lactic acid to glycogen. These data were in direct opposition with the concepts of Margaria et al.

Some physiologists (13,34,44,52) feel that part of the problem with attempting to relate lactic acid production and removal, and oxygen debt accumulation and repayment is the versatility of the organism to remove produced lactic acid by methods other than those requiring recovery oxygen. According to this concept, when considering the production of lactate during exercise, and its removal after exercise, one must also consider its possible fates during exercise, because the blood concentration of lactate at any particular time is a product of the following factors:

(1) the rate of lactate production
(2) the rate of lactate diffusion from the cells to the blood
(3) the rate of lactate removal (56)

Depocas (13) in his research involving the infusion of 14C-lactic acid into dogs exercising at thirty percent of maximum oxygen uptake, found that the rates of lactic acid formation and removal was always greater than at rest. He also reported that seventy-four percent of the lactic

acid produced was converted to carbon dioxide, and that the energy released by the formation of extra lactic acid represented, energetically, less than 0.5% of the extra energy cost of running. Omachi (52), also infusing isotopic lactate into exercising dogs, concluded from his data that the rate of conversion of lactate to carbon dioxide associated with contraction was greater than the increase in the rate of carbon dioxide production, thus inferring that there was an increased utilization of lactate as substrate when the intensity of exercise increased. Jorfeldt (34) using labelled lactate in human subjects, confirmed that there was an exchange of lactate between blood and exercising skeletal muscle, with a simultaneous release and uptake during exercise. The lactate taken up was oxidized in part to carbon dioxide, but other metabolic pathways were claimed to be involved.

Besides skeletal muscles, other organs have been shown to remove lactate from the blood during rest and exercise. Rowell (56) in his measurements of lactate utilization by the liver during exercise, found that in moderate and heavy treadmill exercise, blood lactic acid was time dependent. His data indicated that when exercise was moderate and prolonged, a significant fraction of the lactate produced (fifty-eight percent) was removed by hepatic-splanchnic tissue during the exercise. Gluconeogenesis was the most likely fate of the lactate, as suggested by the low carbon dioxide production and high

oxygen uptake. Lactate uptake has also been measured in the heart (23) and kidney (44) but no data are available with respect to lactate uptake during exercise.

It has been shown that the rate of disappearance of lactate during exercise depends on the intensity, with the maximal removal rate reported to occur at approximately sixty to seventy percent of aerobic capacity (63).

2. Repayment, maintenance and accumulation of oxygen debt

In opposition to Margaria's theory that oxygen debt is accumulated above the threshold level and is maintained at a constant level below the threshold level, research reports hypothesize and, in some cases, show actual oxygen debt data to the contrary.

Schneider et al. (59) maintained that the level of oxygen consumption during work was dependent on the maintenance of a constant oxygen debt as the work was continued, and that the oxygen debt was neither repaid nor increased during work even though the oxygen requirement for the work was far below the man's aerobic capacity. In their research involving exercise at thirty to fifty-five percent of maximum aerobic capacity for three to twenty-five minutes, Schneider found that the oxygen debt remained constant after the third minute of exercise, even though the lactic acid level was decreased after the longer exercise periods. In keeping with the Margaria school of thought, he concluded that perhaps as exercise was prolonged

a change of lactic into alactic debt was occurring. However, no estimations of the components were included in his report to substantiate or refute his hypothesis. Knuttgen (42) reported that after submaximal exercises lasting fifty-five minutes, the measured oxygen debt was greater than the debt after the shorter runs. He reasoned that this was due to a "general body disturbance" involving increases in body temperature and hormonal activity that were not present during the shorter runs. In addition, he found that the alactic component was less during the longer exercise periods and stated that perhaps alactic debt was being repaid during the longer periods. This concept was in disagreement with Schneider's results (which inferred an increased alactic debt over time, if lactic debt was changed into alactic). Even though the components of oxygen debt were estimated in Knuttgen's research, no attempt was made to compare the lactic component of the debt with lactic acid removal.

Rowell (56), in his research on splanchnic removal of lactate during exercise, found that the oxygen debt did not change, even though the lactate concentration was lower during recovery from the longer exercises. He concluded that in moderate exercise, blood lactic acid was time dependent and oxygen debt was not. Rowell also concluded that, in his study, lactic debt was converted into an alactic debt. Actual measurement of the components failed to substantiate this concept.

Other studies investigating the effect of time duration of submaximal exercise on the oxygen debt tended to differ in their findings. Wasserman et al. (68) found no significant differences in the oxygen debt after exercises of ten and twenty minutes at the same intensity. On the other hand, Whipp et al. (73) found a larger total debt after two to three minutes of submaximal exercise than after longer bouts at the same intensity.

In all cases, the reasons put forth for the respective results were based on conjecture. Knuttgen's "general body disturbance concept" might easily have been accepted or rejected by comparing the lactic portion of the debt and the lactic acid removed in the same period. The same might have been done to substantiate or refute Whipp's hypothesis that some of the oxygen debt was repaid during the longer exercise periods.

3. Lactic acid removal versus oxygen debt repayment

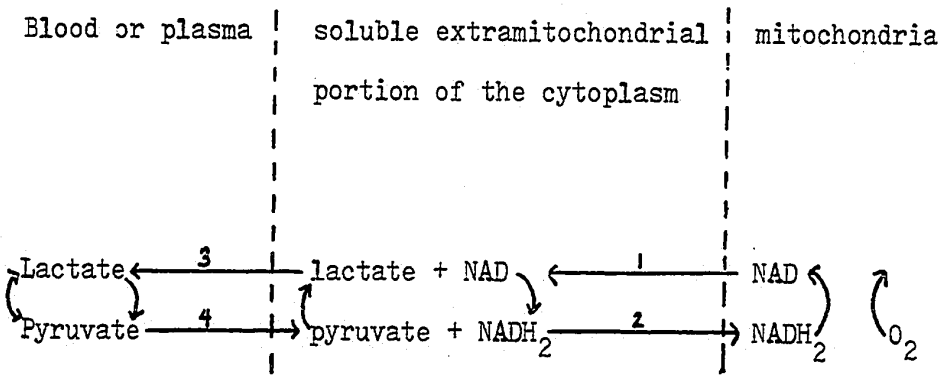
Kayne and Alpert (2, 39) have presented data which, once again, upset some of the preconceived concepts regarding oxygen debt and lactic acid removal. From early research on anaesthetized dog subjected to hypoxia (2), they concluded the relationship between excess oxygen uptake during recovery and lactate removal was coincidental, not causal, due to the fact that lactate was removed without any excess oxygen consumption. In 1964, in an attempt to discover the role of the liver in the oxygen debt repayment

Kayne and Alpert (39) conducted a study in which they hepatectomized dogs and subjected them to forced exercise. Their basis for the research was that if much of the lactate was removed by the liver, then the removal of that organ would alter oxygen debt, if lactate removal was related to oxygen debt. Their conclusions were much the same as from their earlier research; that the relationship between lactate removal and excess oxygen consumption following exercise was coincidental, not causal.

The most significant outcomes of the Kayne and Alpert research were the reasons postulated as to why it was unreasonable to ascribe to the lactate reaction the role of regulator of excess recovery oxygen uptake, which is essentially what one is doing when trying to relate the two. According to their explanation, this would imply a direct cause and effect relationship among the four reactions in Figure 2. In reality, the equilibrium between plasma and intracellular lactate is suspect. Also it is possible that the direct mitochondrial oxidation of external $NADH_2$ is slow or non-existant, and there is some evidence that the mitochondrial membrane in vivo is impermeable to reduced NAD (39). According to Alpert (1),there are systems for shuttling reducing equivalents from cytoplasmic $NADH_2$ to the mitochondrial system, which probably do not include lactate or pyruvate.

More recently, it has been illustrated by Jobsis (40) that the assumption that lactic acid is produced due to an

Fig. 2: Relationship of Lactate, NAD, and Oxygen (39)



insufficiency of oxygen to maintain electron transport is probably false. Mitochondrial NAD-$NADH_2$ was shown to go toward the oxidized state in isolated exercising muscle groups, which indicated an adequate supply of oxygen at the mitochondrial level. At the same time, however, cytoplasmic NAD-$NADH_2$ shifted towards a more reduced state. This indicated that electron transport between the cytoplasm and mitochondrial NAD was inadequate, perhaps due to membrane impermeability, as hypothesized by Kayne and Alpert (39). Thus, lactic acid could be produced in the cytoplasm even in an abundance of oxygen at the mitochondrial level.

According to this scheme, oxygen uptake during recovery, would be related merely to the number of electrons transferred from substrate to molecular oxygen via the respiratory chain, irrespective of lactate formation or removal (39). Boxer and Devlin (8) have confirmed most of these relationships. Therefore, the amount of oxygen

consumed during a specific time interval of recovery and the lactic acid removed during that same interval would not be directly related, as upheld by Margaria et al.

Rowell (56), from his research on the splanchnic removal of lactic acid during prolonged submaximal exercise, presented four factors which he felt determined the correlation between the quantity of lactate in the body at the time of recovery and the oxygen debt:

(1) the duration of exercise
(2) blood flow to "resting" tissues
(3) the capacity of resting tissues to substitute lactate for other substrate
(4) the quantity of lactate produced in relation to the metabolic rate of resting tissues.

Rowell also stated that it was difficult to see how a stoichiometric relationship could ever exist between oxygen debt and lactic acid once the latter had diffused from working muscle and tissues which readily convert it into glucose or oxidize it in substitution for other substrate (56). The research presented in this section based on histological as well as physiological study provides or infers concepts of oxygen debt and lactic acid removal which may be directly opposed to the concepts originally constructed by Margaria and co-workers. However, the question still remains as to whether Margaria's concepts and the ones than appear to be contradictory to them are,

in fact, reconcilable.

Conclusions from the Literature

Review of the literature indicates that at least two theories exist at the present time regarding oxygen debt and the corresponding lactic acid removal following submaximal exercise of various intensities and durations. In particular, diverse opinions exist as to whether oxygen debt is repaid, maintained, or accumulated during submaximal exercise, and to whether lactic acid removal during the recovery period is causally or coincidentally related to the excess oxygen consumption occurring at the same time. Despite the abundance of literature claiming to refute the Margaria *et al.* theory of oxygen debt, in no case has the theory been adequately tested by comparing the lactic acid removed during the recovery period to the estimated lactic portion of the oxygen debt repaid during the same period. With the results of such a research attempt, one would undoubtedly be justified in either accepting or refuting the Margaria *et al.* theory.

CHAPTER III

METHODOLOGY

Testing Procedures

Twelve male subjects, who were involved in regular training for competitive running of at least two miles, were randomly assigned into three test groups, designated Group A, Group B, and Group C. Each group was intended to represent a specific intensity of maximum oxygen uptake; fifty percent for Group A, sixty-five percent for Group B, and eighty percent for Group C.

Costill (11) presented data which indicated that well-trained distance runners were able to maintain their level of energy expenditure at a particular percentage of their maximum oxygen capacity, with no significant changes, for up to three hours. Knuttgen (42), reporting on oxygen debt following exercise of varied duration, found a steady elevation in oxygen uptake values during the longer exercise periods after testing untrained normal individuals. Due to the urgency, in the present study, that exercise uptake levels remain stable during the longer runs in order to compare the corresponding oxygen debt parameters, individuals accustomed to prolonged running were used as subjects in the study.

Preliminary Testing Procedures

Before the actual submaximal runs were conducted, each subject underwent a series of preliminary tests to determine maximum aerobic capacity and to determine the treadmill speed to be used in the later runs which would elicit the desired submaximal intensity of approximately fifty percent (Group A), sixty-five percent (Group B) and eighty percent (Group C).

To determine the maximum oxygen uptake, each subject performed duplicate modified Taylor maximum aerobic capacity tests. The criteria for maximum oxygen uptake was that the difference between any two levels of the progressive test be less than one hundred fifty millilitres of oxygen uptake per minute (65). The largest of the two values found on the tests was taken as the subject's maximum oxygen uptake.

To determine the treadmill speed that would be used in the subsequent runs to elicit the desired submaximal oxygen uptake, each subject was run on a twenty-minute progressive treadmill run, in which the speed was increased every five minutes. Oxygen uptake measurements were taken at each treadmill speed, and these values were plotted against speed to obtain a linear relationship. The desired oxygen uptake for the submaximal runs was then extrapolated to find the desired treadmill speed. In a later test, the subject was run for five minutes at the extrapolated treadmill speed, and the speed was adjusted according to whether

or not it elicited the approximate desired submaximal oxygen uptake.

Submaximal Testing Procedures

Before reporting for the submaximal tests, the subjects were instructed to ingest no food for at least three hours before the test, and to engage in no strenuous activity the day of the test. These factors have been shown to possibly affect resting lactate levels (41,42,50).

The subject sat on a chair, on the treadmill apron, for ten to fifteen minutes before the test, during which time pre-exercise expired air samples and blood samples were collected for the later determination of resting oxygen uptake and blood lactic acid concentration, respectively. The subject then ran for five, fifteen or thirty minutes at the treadmill speed that was found in the preliminary tests to elicit the desired submaximal oxygen uptake. Thirty second oxygen uptake measurements were taken at the beginning of the fifth minute of the five minute run, at the beginning of the fifth, tenth and fifteenth minutes of the fifteen minute run, and at the beginning of the fifth, tenth, fifteenth and thirtieth minutes of the thirty minute run.

At the end of the run, the treadmill was stopped, and a chair placed on the treadmill apron. The subject was instructed to sit and relax. Continuous measurements of oxygen uptake were started at the end of the run and continued for thirty minutes. Blood samples were taken at

the beginning of the third, sixth, ninth, nineteenth and twenty-ninth minute of recovery, and later analyzed for lactic acid content.

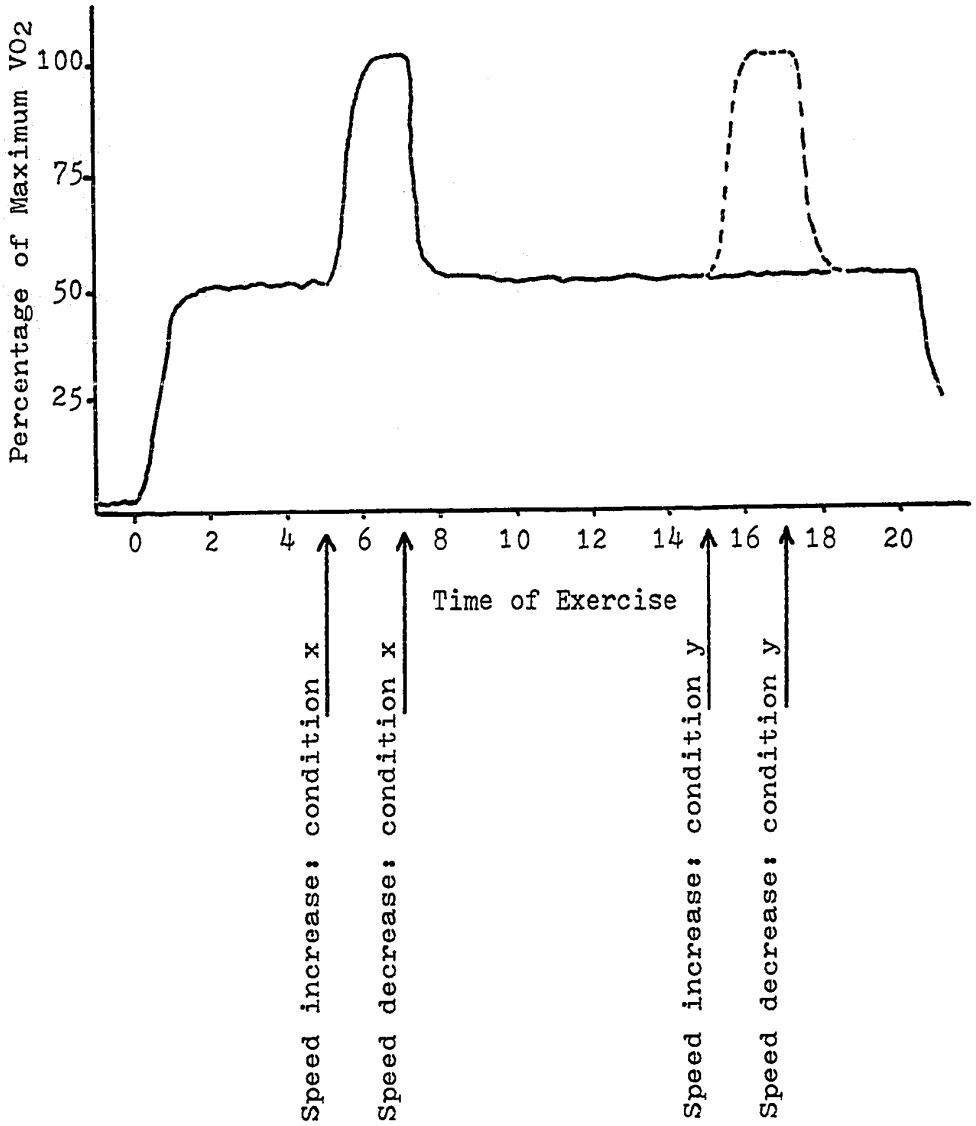
One subject from each of the three intensity groups voluntarily took part in two additional submaximal runs. These were two twenty minute runs at the same intensity as the others, but with an added increase in intensity at one point during the run (see Figure 3). The added intensity was an increase in speed to a pace that could be tolerated no longer than two and one-half minutes. During one of the runs, the increase in speed was at the beginning of the fifth minute of the submaximal run and, after two minutes at this increased speed, the speed was decreased to the previous submaximal treadmill speed. During the second run, the increase in speed occurred at the beginning of the fifteenth minute of the run. In both cases, oxygen uptake was measured during the last thirty seconds of the increased intensity to insure that the oxygen uptake at this point was near maximum (15). As in the other runs, recovery oxygen uptake was measured continuously, and blood samples were taken for lactic acid determination.

Measurement Techniques

1. Oxygen Uptake

Oxygen uptake was measured using the open circuit technique (10). A Collins High-Speed Triple-"J" valve and a set of 2½ I.D. corrugated plastic hoses were used to partition inspired and expired gases. The inspired volume

Figure 3 :--Sample Protocol for Special Condition Run



was measured by a Parkinson-Cowan Dry Gas Meter and, by means of a potentiometer, was relayed and recorded on a Hewlett-Packard Eight Channel Recorder (model 1060A). All gas volumes were corrected to STPD. For all pre-exercise and exercise measurements, samples of expired air were drawn into well-lubricated 50 cc. Luer-lok syringes from a collecting manifold through which expired air was being exchanged from a mixing cannister. The samples were analyzed for carbon dioxide and oxygen content on a Llyod Gallenkamp gas analyzer, with random duplicate samples taken to insure adequate consistency.

In order that continuous measurement of oxygen uptake be taken during the recovery period following the submaximal runs, continuous carbon dioxide, oxygen and inspired volume measurements were recorded from the end of the run to the end of the thirty minute recovery period. Carbon dioxide was monitored by a Godart Capnograph (model #145) and oxygen was analyzed by a Westinghouse Pulmonary Function Oxygen Monitor (model #211). These values were continuously recorded on the Hewlett-Packard recorder. The Westinghouse and Godart analyzers were calibrated before every test with reference gas concentrations that spanned the analysis range.

Lactic Acid Concentration Determination

During the pre-exercise period, and at the specified periods during recovery, blood samples were taken from the fingertip of a pre-warmed hyperemic hand, a method which enabled accurate estimation of arterial blood lactate

concentration (42). At the time of sampling, the hand was withdrawn from the water, the fingertip was dried and sterilized with alcohol, and the fingertip was punctured with a steel disposable lancet. The first drop was wiped away, and .05 ml. of blood was collected in a disposable, graduated glass pipette. The blood sample was then blown into a test tube containing 1.9 ml. of a five percent trichloroacetic acid solution, the solution in the test tube was immediately mixed and put into cold storage, to insure minimum change in lactic acid content (28).

Analysis of blood lactate concentration was made according to the method of Barker and Summerson (6) as modified by Strom (64). On each day of testing, a set of known standard lactic acid concentrations was made and put through the analysis procedures with the blood samples. The standard concentrations in mg% were plotted against the absorbance readings from the Bausch and Lomb Spectrophotometer. From the linear relationship that resulted, the absorbance readings of the blood samples were extrapolated to obtain the blood lactate concentrations (unpublished manuscript, University of Windsor).

Plotting the Oxygen Debt Curves

From the recording of the continuous oxygen, carbon dioxide and inspired volume measurements taken throughout the recovery period, oxygen uptake values were calculated, by an IBM 360/50 computer, at fifteen second intervals for the first four minutes, at thirty second intervals from the

fourth to the tenth minute, at sixty second intervals from the tenth to the fifteenth minute, and for ninety second intervals from the fifteenth minute to the end of the thirty minute recovery period.

The mean oxygen uptake values of each recovery interval for each duration and intensity of exercise were plotted on linear graph paper to determine the asymptote[1]. The mean oxygen uptake values minus the asymptotic values were plotted on tri-phase semilogarithmic paper and the asymptotic value adjusted to improve the linearity of the lactic component, if necessary. The alactic and lactic components were estimated by inspection, a method which has been used previously (25,26). Then, using Henry's formula; $a_1e^{-k_1t} + a_2e^{-k_2t}$ for the oxygen debt curve, the values of k_1, and k_2 were calculated. The alactic ($^{a}1/k_1$) and the lactic ($^{a}2/k_2$) components of the oxygen debt, as well as the total oxygen debt ($^{a}1/k_1 + {}^{a}2/k_2$) were derived.

Comparison of Lactic Acid and Oxygen Debt Data

In order to relate the lactic acid removed during recovery with the lactic portion of the oxygen debt repaid, during the same period, the lactic acid removed was converted to oxygen equivalents. Total body lactate was

[1]The asymptote is that point at which the curve no longer assumes a continuous decrease toward resting levels. This measure was used as a base line in lieu of pre- or post-exercise oxygen uptake in order to eliminate the inclusion of factors other than lactic component in the lactic portion of the debt.

calculated using the equation proposed by Margaria (48):

$$LA/kg = \frac{0.61}{0.80} \cdot LA_b$$

in which total body lactate in grams per kilogram of body weight (LA/kg) is related to blood lactate in grams per litre of blood (LA_b) as indicated, assuming that total body water is sixty-one percent of the body weight and the blood is eighty percent water (48).

This value, in grams per kilogram of body weight, was then converted to grams by multiplying the value by the mean weight of the group in kilograms. The resulting value was converted to oxygen equivalents required to remove the lactate by multiplying by .1244, which is the amount of oxygen necessary to remove one gram of lactate. This last computation is from Hermansen (29), who states that one mole (22.4 litres) of oxygen is necessary to remove two moles (180 grams) of lactate.

Statistical Procedures

In order to test the hypotheses, the following statistical procedures were employed, using $\alpha = .05$ as the level of significance.

1. The exercise oxygen uptake measurements during the fifteen and thirty minute runs, and at the same time interval during different durations of running for each group, were each subjected to a one-way analysis of variance for repeated measures (74) to determine inter

and intra-run variation within each group.

2. The individual actual oxygen debt measurements for each group were subjected to one-way analysis of variance for repeated measures to determine the effect of duration of exercise on the magnitude of the oxygen debt.

3. The actual oxygen debt measurements from minute six to minute twenty-nine of recovery for each group were subjected to one-way analysis of variance for repeated measures to determine the effect of duration of exercise on the lactic component of the oxygen debt[1].

4. A t-test for correlated observations (17) was used to test for significant differences between the oxygen debt repaid from minute six to minute twenty-nine of recovery and the lactic acid removed during the same interval, expressed in oxygen equivalents, for each intensity group and duration of exercise.

In an attempt to further clarify the relationships tested statistically, the following procedures were employed:

5. The alactic components of oxygen debt for each intensity group were examined for possible treads, resulting from the duration of exercise.

6. The actual oxygen debt measurements, and the

[1]The basis for the use of the actual oxygen debt measurements during this period as representative of the lactic component is outlined in Chapter IV.

actual oxygen debt measurements from minute six to minute nineteen of recovery from the two special condition runs at each level of intensity, were examined to find if there were possible changes in these parameters resulting from changing the time during steady state running, at which a maximal bout of exercise was presented. In addition, the relationship between lactic acid removal and oxygen debt repayment during the same period were examined in relation to the statistical results found between these parameters after the regular submaximal runs.

CHAPTER IV

RESULTS

The descriptive data of each subject, as well as the means and standard deviations for the characteristics of the subjects within each group, are presented in Table 1. Analysis of variance for unequal group sizes performed on each characteristic revealed no significant differences between groups at the .05 level of confidence (Tables 2,3, and 4).

In order that the oxygen debt measurements of the three durations of exercise be comparable within each intensity group, the exercise oxygen uptake data collected during exercise was subjected to analysis of variance to determine the significance of between-run and within-run variation. In addition, in order to insure that the alactic components of the debt were comparable, statistical procedures were employed to validate the theoretical oxygen debt curves.

Exercise Oxygen Uptake

The mean exercise oxygen uptake measurements of Group A, Group B, and Group C were 2.36 litres/min., 2.51 litres/min., and 3.28 litres/min., respectively (Table 5). These values, expressed as percentages of the maximum oxygen uptaken for each group, were 56.1%, 63.5% and 79.6%. Analysis of variance for unequal group sizes indicated a significant

TABLE 1

CHARACTERISTICS OF SUBJECTS AND ASSIGNMENT INTO GROUPS

Group A: 50% of VO_2 Maximum				
Subject	Age (yrs.)	Wt.(kg.)	VO_2 max. (l./min.)	Submax. VO_2 (l./min.)
A.K.	19.0	70.45	3.93	1.97
B.G.	41.0	80.00	4.21	2.11
P.M.	21.0	76.59	4.45	2.23
$\bar{X}$	27.0	75.68	4.20	2.10
S.D.	± 9.5	± 3.95	± .21	± .11

Group B: 65% of VO_2 Maximum				
Subject	Age (yrs.)	Wt.(kg.)	VO_2 max. (l./min.)	Submax. VO_2 (l./min.)
B.F.	21.0	74.55	4.41	2.87
G.P.	39.0	69.55	3.31	2.15
J.S.	40.0	100.00	3.95	2.57
D.B.	21.0	77.27	4.13	2.68
$\bar{X}$	30.3	80.34	3.95	2.57
S.D.	± 9.1	±11.64	± .40	± .27

Group C: 80 % of VO_2 Maximum				
Subject	Age (yrs.)	Wt.(kg.)	VO_2 max. (l./min.)	Submax. VO_2 (l./min.)
G.M.	30.0	74.55	4.70	3.76
H.B.	26.0	60.45	3.82	3.06
J.M.	19.0	65.00	3.74	2.99
K.C.	19.0	72.72	4.20	3.36
$\bar{X}$	23.0	68.18	4.12	3.29
S.D.	± 4.7	± 6.23	± .38	± .34

TABLE 2

ANALYSIS OF VARIANCE OF SUBJECT AGE

Source of Variance	S.S.	df	M.S.	F.
Treatments	91.160	2	45.58	.501*
Error	727.750	8	90.97	
Total	818.910	10		

*$F_{.95}(2,8) < 4.46$

TABLE 3

ANALYSIS OF VARIANCE OF SUBJECT
MAXIMUM OXYGEN UPTAKE

Source of Variance	S.S.	df	M.S.	F.
Treatments	.11	2	.06	.353**
Error	1.36	8	.17	
Total	1.47	10		

**$F_{.95}(2,8) < 4.46$

TABLE 4

ANALYSIS OF VARIANCE OF SUBJECT WEIGHT

Source of Variance	S.S.	df	M.S.	F.
Treatments	300.25	2	150.13	1.66*
Error	723.78	8	90.47	
Total	1024.03	10		

*$F_{.95}(2,8) < 4.46$

TABLE 5

MEAN EXERCISE OXYGEN UPTAKE MEASUREMENTS

Group A		Group B		Group C	
Subj.	O_2 Uptake (l./min.)	Subj.	O_2 Uptake (l./min.)	Subj.	O_2 Uptake (l./min.)
B.G.	2.45	G.P.	2.37	H.B.	3.22
P.M.	2.22	J.S.	2.28	G.M.	3.43
A.K.	2.40	B.F.	2.81	J.M.	2.83
		D.B.	2.57	K.C.	3.62
$\bar{X}$	2.36		2.51		3.28

difference among the percentages of maximum oxygen uptake of the groups at the .05 level (Table 6). The subsequent Newman-Keuls procedure revealed that Group C was significantly different from Group A and Group B on this parameter, while Group A and Group B were not significantly different at the .05 level (Table 7).

For each intensity group, analysis of variance of the minute four, minute nine, and minute fourteen oxygen uptake measures among the three durations of exercise revealed no significant differences at the .05 level of confidence (Tables 8 to 17), with one exception. In the Group A oxygen uptake measures, a significant difference was indicated by the analysis of variance among those measurements taken starting at minute four of the runs (Table 8). The subsequent Newman-Keuls procedure revealed that these values were all different from one another (Table 9).

Analysis of variance of the oxygen uptake measurements taken at intervals during the fifteen and thirty minute runs indicated no significant differences in these measurements for all groups (Tables 18 to 23).

Analysis of variance of the oxygen uptake measurements taken during the last minute of exercise revealed no significant differences at the .05 level among the three durations of exercise within each group (Tables 24,25 and 26).

The analysis of variance of the exercise oxygen uptake measurements for each group indicated no intra- or inter-run variation at the .05 level, with one exception, thus, in most cases, warranting comparison of oxygen debt

TABLE 6

ANALYSIS OF VARIANCE OF MEAN PERCENTAGES
OF MAXIMUM OXYGEN UPTAKE

Source of Variance	S.S.	df	M.S.	F.
Treatments	1028.07	2	514.04	14.06*
Error	292.37	8	36.55	
Total	1320.44	10		

*$F_{.95}(2,8) > 4.46$

TABLE 7

NEWMAN-KEULS MEANS TEST OF PERCENTAGES
OF MAXIMUM OXYGEN UPTAKE

Groups		A	B	C
	Means	56.38	63.82	79.78
A	56.38	----	7.44**	23.40**
B	63.82	----	----	15.96**
C	79.78	----	----	----
$q_{.95}(r,8)$			3.26	4.04
MSerror/n $q_{.95}(r,8)$			10.38	12.86

TABLE 8

ANALYSIS OF VARIANCE OF GROUP A
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOUR (N=3)

Source of Variance	S.S.	df	M.S.	F.
Between people	.254	2		
Within people	.162	6		
treatment	.127	2	.0635	7.26*
residual	.035	4	.0088	
Total	.416	8		

*$F_{.95}(2,4) > 6.94$

TABLE 9

NEWMAN-KEULS MEANS TEST OF GROUP A
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOUR (N=3)

Runs		5 min.	30 min.	15 min.
	Totals	6.64	6.95	7.49
5	6.64	----	.31**	.85**
30	6.95	----	----	.54**
15	7.49	----	----	----
$q_{.95}(r,4)$		.393	.504	
nMSres $q_{.95}(r,4)$		.064	.082	

TABLE 10

ANALYSIS OF VARIANCE OF GROUP A
EXERCISE OXYGEN UPTAKE DATA
MINUTE NINE (N=3)

Source of Variance	S.S.	df	M.S.	F.
Between people	.068	2		
Within people	.119	3		
treatment	.062	1	.062	2.138*
residual	.057	2	.029	
total	.187	5		

*$F_{.95}(1,2) < 18.5$

TABLE 11

ANALYSIS OF VARIANCE OF GROUP A
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOURTEEN (N=3)

Source of Variance	S.S.	df	M.S.	F.
Between people	.024	2		
Within people	.028	3		
treatment	.010	1	.010	1.111**
residual	.018	2	.009	
total	.052	5		

**$F_{.95}(1,2) < 18.5$

TABLE 12

ANALYSIS OF VARIANCE OF GROUP B
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOUR (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	1.086	3		
Within people	.280	8		
treatment	.010	2	.005	.111*
residual	.270	6	.045	
total	1.366	11		

*$F_{.95}(2,6) < 5.14$

TABLE 13

ANALYSIS OF VARIANCE OF GROUP B
EXERCISE OXYGEN UPTAKE DATA
MINUTE NINE (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.341	3		
Within people	.624	4		
treatment	.042	1	.042	.216**
residual	.582	3	.194	
total	.965	7		

**$F_{.95}(1,3) < 10.1$

TABLE 14

ANALYSIS OF VARIANCE OF GROUP B
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOURTEEN (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.371	3		
Within people	.112	4		
treatment	.004	1	.004	.111*
residual	.108	3	.036	
total	.483	7		

*$F_{.95}(1,3) < 10.1$

TABLE 15

ANALYSIS OF VARIANCE OF GROUP C
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOUR (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.174	3		
Within people	.705	8		
treatment	.146	2	.073	.261**
residual	.559	6	.280	
total	.879	11		

**$F_{.95}(2,6) < 5.14$

TABLE 16

ANALYSIS OF VARIANCE OF GROUP C
EXERCISE OXYGEN UPTAKE DATA
MINUTE NINE (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.394	3		
Within people	.144	4		
treatment	.037	1	.037	1.028*
residual	.107	3	.036	
total	.538	7		

*$F_{.95}(1,3) < 10.1$

TABLE 17

ANALYSIS OF VARIANCE OF GROUP C
EXERCISE OXYGEN UPTAKE DATA
MINUTE FOURTEEN (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.197	3		
Within people	.259	4		
treatment	.004	1	.004	.047**
residual	.255	3	.085	
total	.456	7		

**$F_{.95}(1,3) < 10.1$

TABLE 18

ANALYSIS OF VARIANCE OF GROUP A
EXERCISE OXYGEN UPTAKE DATA
FIFTEEN MINUTE RUN (N=3)

Source of Variance	S.S.	df	M.S.	F.
Between people	.091	2		
Within people	.044	6		
treatment	.009	2	.0045	.257*
residual	.035	4	.0175	
total	.135	8		

*$F_{.95}(2,4) < 6.94$

TABLE 19

ANALYSIS OF VARIANCE OF GROUP A
EXERCISE OXYGEN UPTAKE DATA
THIRTY MINUTE RUN (N=3)

Source of Variance	S.S.	df	M.S.	F.
Between people	.102	2		
Within people	.229	9		
treatment	.056	3	.019	.655**
residual	.173	6	.029	
total	.331	11		

**$F_{.95}(3,6) < 4.76$

TABLE 20

ANALYSIS OF VARIANCE OF GROUP B
EXERCISE OXYGEN UPTAKE DATA
FIFTEEN MINUTE RUN (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.217	3		
Within people	.465	8		
treatment	.016	2	.008	.107*
residual	.449	6	.075	
total	.682	11		

*$F_{.95}(2,6) < 5.14$

TABLE 21

ANALYSIS OF VARIANCE OF GROUP B
EXERCISE OXYGEN UPTAKE DATA
THIRTY MINUTE RUN (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	1.150	3		
Within people	.650	12		
treatment	.094	3	.031	.500**
residual	.555	9	.062	
total	1.800	15		

**$F_{.95}(3,9) < 3.86$

TABLE 22

ANALYSIS OF VARIANCE OF GROUP C
EXERCISE OXYGEN UPTAKE DATA
FIFTEEN MINUTE RUN (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.640	3		
Within people	.075	8		
treatment	.015	2	.008	.800*
residual	.060	6	.010	
total	.715	11		

*$F_{.95}(2,6) < 5.14$

TABLE 23

ANALYSIS OF VARIANCE OF GROUP C
EXERCISE OXYGEN UPTAKE DATA
THIRTY MINUTE RUN (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.322	3		
Within people	.773	12		
treatment	.106	3	.035	.473**
residual	.667	9	.074	
total	1.095	15		

**$F_{.95}(3,9) < 3.86$

TABLE 24

ANALYSIS OF VARIANCE OF GROUP A
LAST MINUTE OXYGEN UPTAKE (N=3)

Source of Variance	S.S.	df	M.S.	F.
Between people	.044	2		
Within people	.133	6		
treatment	.086	2	.043	3.58*
residual	.047	4	.012	
total	.177	8		

*$F_{.95}(2,4) < 6.94$

TABLE 25

ANALYSIS OF VARIANCE OF GROUP B
LAST MINUTE OXYGEN UPTAKE (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.563	3		
Within people	.655	8		
treatment	.022	2	.011	.104**
residual	.633	6	.106	
total	1.218	11		

**$F_{.95}(2,6) < 5.14$

TABLE 26

ANALYSIS OF VARIANCE OF GROUP C
LAST MINUTE OXYGEN UPTAKE (N=4)

Source of Variance	S.S.	df	M.S.	F.
Between people	.151	3		
Within people	.380	8		
treatment	.014	2	.007	.115*
residual	.366	6	.061	
total	.531	11		

*$F_{.95}(2,6) < 5.14$

parameters after the three durations of running within each group.

Validation of the Theoretical Oxygen Debt Curves

Total Oxygen Debt

The actual total oxygen debt measurements and the corresponding theoretical values are shown in Table 27. A correlated t-test performed on the actual total oxygen debt versus the oxygen debt as derived from the theoretical curve yielded no significant differences between the two measures at the .05 level for the three pairs of group data (Table 28).

Oxygen Debt from Minute 6 to Minute 29 of Recovery

Examination of the theoretical curve (see example in Figure 4) revealed that, after the sixth minute of recovery, the remaining oxygen debt consisted entirely of lactic component. Therefore, the accuracy of the estimation of that component from the sixth to the twenty-ninth minute of recovery, and of the total lactic component, could be validated by comparing that portion of the component with the actual oxygen debt during the assocated period. The individual actual oxygen debt measurements from minute six to twenty-nine are shown in Table 29, and the corresponding measurements from the theoretical curves are shown in Table 30. Correlated t-tests performed between the pairs of oxygen debt values within each group yielded a significant t-ratio between the two measurements for Group A, thus

TABLE 27

COMPARISON OF ACTUAL TOTAL OXYGEN DEBT MEANS VERSUS THEORETICAL OXYGEN DEBT MEANS FOR EACH GROUP AND DURATION

Group	5 minutes		15 minutes		30 minutes	
	Act.*	Theo.**	Act.	Theo.	Act.	Theo.
A	3.33	3.18	3.37	3.60	3.57	3.71
B	4.09	4.15	4.02	3.89	4.12	4.09
C	4.51	4.49	5.07	5.19	5.83	5.94

* actual total oxygen debt
** theoretical total oxygen debt

TABLE 28

CORRELATED t-TESTS OF ACTUAL TOTAL OXYGEN DEBT VERSUS CORRESPONDING THEORETICAL OXYGEN DEBT

	Group A	Group B	Group C
t-ratio	.702*	.606*	.045*

*$t_{.95}(2) < 4.303$

Figure 4:--Example of Theoretical Curve

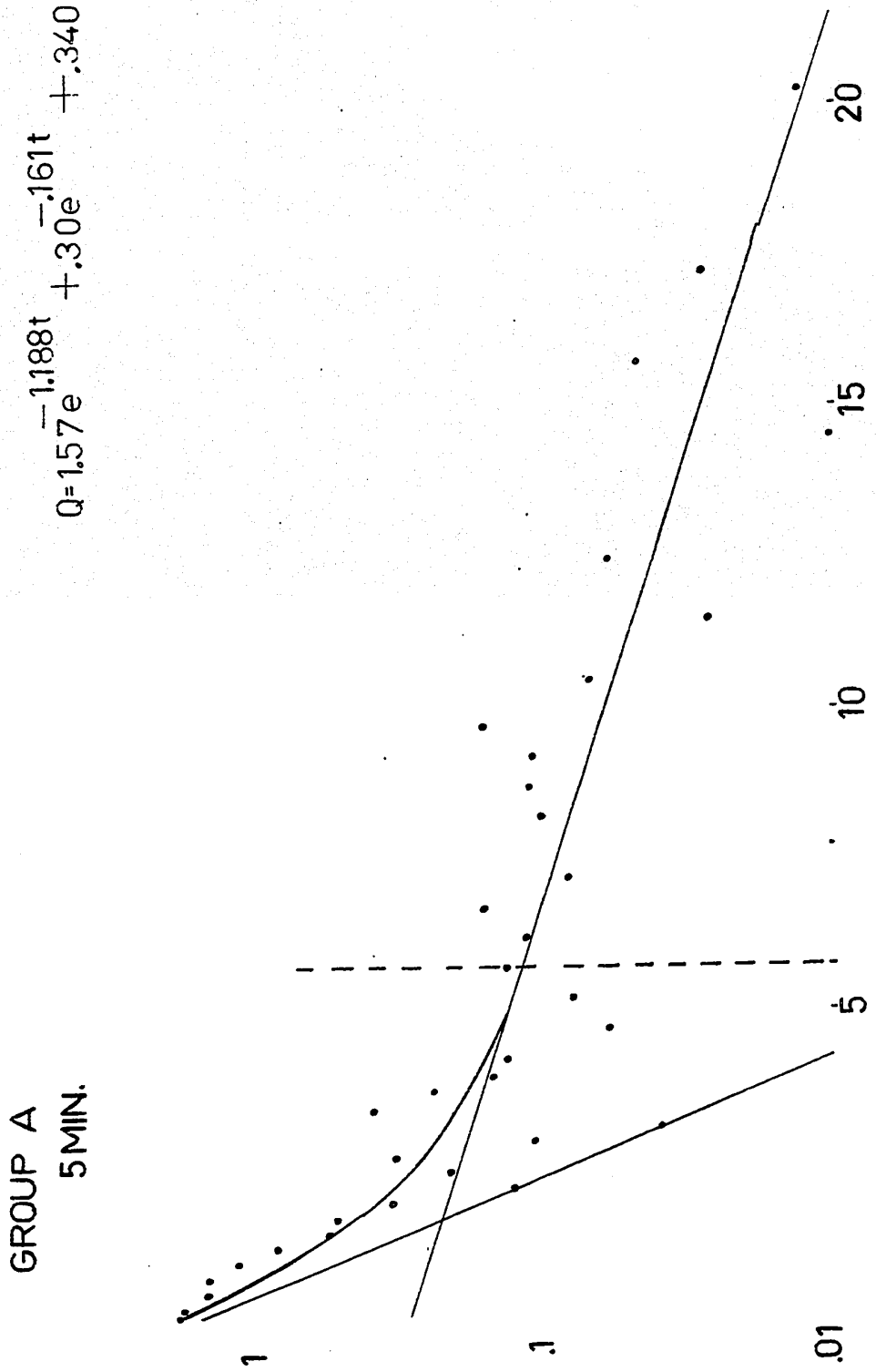


TABLE 29

INDIVIDUAL ACTUAL OXYGEN DEBT FROM MINUTE 6 TO MINUTE 29 OF RECOVERY

Group A

Subjects	5 Minute	15 Minute	30 Minute
B.G.	.955	.550	.985
P.M.	.840	.488	.155
A.K.	.700	1.315	.805
$\bar{X}$	.832	.783	.648

Group B

Subjects	5 Minute	15 Minute	30 Minute
D.B.	1.550	1.355	1.050
G.P.	.855	.655	.485
J.S.	1.060	1.245	.780
B.F.	1.040	1.070	.665
$\bar{X}$	1.127	1.084	.745

Group C

Subjects	5 Minute	15 Minute	30 Minute
G.M.	1.280	1.755	2.430
K.C.	1.040	.515	1.580
H.B.	.500	.698	.940
J.M.	.390	1.555	.910
$\bar{X}$	.803	1.130	1.465

TABLE 30

THEORETICAL OXYGEN DEBT FROM
MINUTE 6 TO MINUTE 29

Group	Duration	A_{2_1}*	A_{2_2}**	k_2***	$\frac{A_{2_1}-A_{2_2}}{k_2}$****
A	5	.116	.003	.161	.701
	15	.113	.003	.163	.675
	30	.112	,002	.184	.598
B	5	.184	.005	.158	1.132
	15	.139	.002	.126	1.087
	30	.143	.002	.190	.742
C	5	.153	.002	.188	.803
	15	.176	.003	.163	1.061
	30	.238	,001	.163	1.460

* the amount of the lactic component, as measured from the theoretical curve, at minute six of recovery.

** the amount of the lactic component, as measured from the theoretical curve, at minute twenty-nine of recovery.

*** the lactic recovery velocity constant.

**** the amount of lactic component between minute six and minute twenty-nine of recovery.

TABLE 31

CORRELATED t-TESTS OF ACTUAL OXYGEN DEBT FROM
MINUTE 6 TO MINUTE 29 VERSUS THE
CORRESPONDING THEORETICAL OXYGEN DEBT

	Group A	Group B	Group C
t-ratio	4.374*****	.154******	1.087******

***** $t_{.95}(2) > 4.303$

****** $t_{.95}(2) < 4.303$

opening to question the validity of the theoretical oxygen debt values for this group. However, the correlated t-tests performed on the corresponding data of Group B and Group C yielded no significant differences at the .05 level (Table 31).

As a result of these validation procedures, it was concluded that the theoretical oxygen debt curves and their respective components were estimated with sufficient accuracy, except in the case of the Group A data, to warrant comparison of the alactic components within each group. Subsequent comparisons among the alactic components of Group A were drawn with the understanding that at least one of the three components of the three theoretical curves was inaccurate.

The Actual Total Oxygen Debt

The individual measurements of the actual total oxygen debt are presented in Table 32. One-way analysis of variance for repeated measures of the measurements for each intensity group indicated no significant differences at the .05 level of confidence (Tables 33,34 and 35).

The Actual Oxygen Debt from Minute 6 to Minute 29

Since the portion of the lactic component of the oxygen debt for a specific time interval in the same proportion to the entire lactic component for all curves (by the Law of Proportionality (75)), the actual oxygen debt measurements during minute six to minute twenty-nine of recovery were subjected to analysis of variance to determine the effect

TABLE 32

ACTUAL TOTAL OXYGEN DEBT

Group A

Subjects	5 Minute	15 Minute	30 Minute
A.K.	2.84	4.04	4.33
P.M.	3.44	2.54	2.76
B.G.	3.71	3.54	3.62
$\bar{X}$	3.33	3.37	3.57

Group B

Subjects	5 Minute	15 Minute	30 Minute
B.F.	4.16	4.07	3.82
D.B.	4.62	4.90	5.26
J.S.	3.43	3.92	3.25
G.P.	4.15	3.18	4.14
$\bar{X}$	4.09	4.02	4.12

Group C

Subjects	5 Minute	15 Minute	30 Minute
G.M.	4.98	6.26	7.28
K.C.	5.70	4.31	5.94
H.B.	3.59	3.88	4.44
J.M.	3.75	5.82	5.69
$\bar{X}$	4.51	5.07	5.83

TABLE 33

ANALYSIS OF VARIANCE OF ACTUAL TOTAL OXYGEN DEBT AFTER THREE DURATIONS OF EXERCISE: GROUP A

Source of Variance		S.S.	df	M.S.	F.
Between people		1.196	2		
Within people		1.700	6		
treatment	.10		2	.05	.125*
residual	1.60		4	.40	
total		2.896	8		

*$F_{.95}(2,4) < 6.94$

TABLE 34

ANALYSIS OF VARIANCE OF ACTUAL TOTAL OXYGEN DEBT AFTER THREE DURATIONS OF EXERCISE: GROUP B

Source of Variance		S.S.	df	M.S.	F.
Between people		3.256	3		
Within people		1.129	8		
treatments	.021		2	.011	.060**
residual	1.108		6	.184	
total		4.385	11		

**$F_{.95}(2,6) < 5.14$

TABLE 35

ANALYSIS OF VARIANCE OF ACTUAL TOTAL OXYGEN DEBT AFTER THREE DURATIONS OF EXERCISE: GROUP C

Source of Variance	S.S.	df	M.S.	F.
Between people	7.41	3		
Within people	7.26	8		
treatments	3.58	2	1.79	2.93*
residual	3.68	6	.61	
total	14.67	11		

*$F_{.95}(2,6) < 5.14$

TABLE 36

ANALYSIS OF VARIANCE OF ACTUAL OXYGEN DEBT FROM MINUTE 6 TO MINUTE 29 OF RECOVERY: GROUP A

Source of Variance	S.S.	df	M.S.	F.
Between people	.325	2		
Within people	.569	6		
treatments	.054	2	.027	.211**
residual	.515	4	.128	
total	.894	8		

**$F_{.95}(2,4) < 6.94$

of duration of exercise on this component. The graph in Figure 5 suggests a steady decline in the amount of this component in the results of the Group A and B data, and a gradual increase over time in the component for the Group C measures. The Group A data showed a steady decline in the lactic component to seventy-seven percent of its original value after thirty minutes of exercise. Similarly, the lactic component of the oxygen debt after thirty minutes was sixty-six percent of the component measured after the five minutes of running for Group B. On the other hand, the Group C lactic component was increased after the thirty minute run by 82.4% of the component after the five minute run, suggesting a significant increase over time.

Analysis of variance revealed a significant F-ratio among the Group B lactic component figures (Table 37). The subsequent Newman-Keuls means tests confirmed that the lactic component of the debt was significantly less following the thirty minute run than following the fifteen and five minute runs, with no significant difference revealed between the latter two (Table 38). Analysis of variance of the corresponding data for Group A (Table 36) and Group C (Table 39) indicated no significant differences among the three durations of exercise at the .05 level of confidence.

The Group C data, which appeared to include significant differences among the mean values (Figure 5) contained a considerable degree of within-individual variance not attributed to the treatment effect (Table 39).

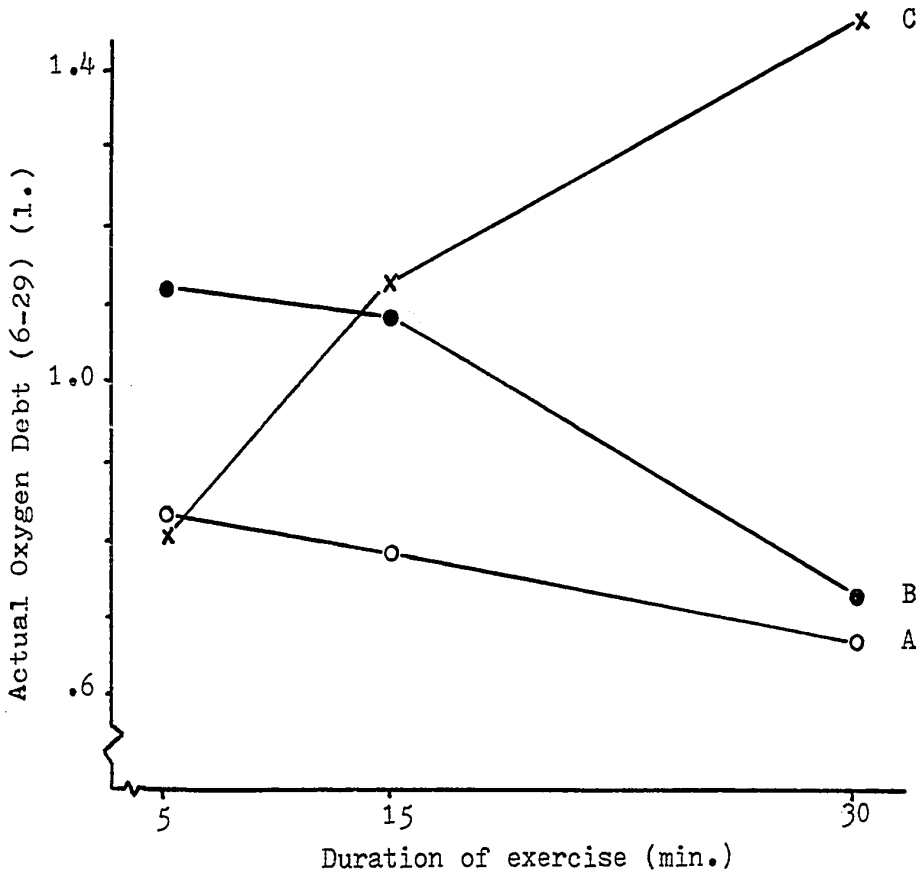


Figure 5:--Actual Oxygen Debt (min. 6 to 29) vs. Duration of Exercise

TABLE 37

ANALYSIS OF VARIANCE OF ACTUAL OXYGEN DEBT
FROM MINUTE 6 TO MINUTE 29
OF RECOVERY: GROUP B

Source of Variance	S.S.	df	M.S.	F.
Between people	.657	3		
Within people	.407	8		
treatment	.347	2	.174	17.4*
residual	.060	6	.010	
total	1.064	11		

*$F_{.95}(2,6) > 5.15$

TABLE 38

NEWMAN-KEULS MEANS TEST OF ACTUAL OXYGEN DEBT
MEANS FROM MINUTE 6 TO MINUTE 29
OF RECOVERY: GROUP B

Duration		5	15	30
	Totals	4.505	4.325	2.980
5	4.505	-----	.180	1.525**
15	4.325	-----	-----	1.345**
30	2.980	-----	-----	-----
$q_{.95}(r,6)$		3.46	4.34	
nMSres $q_{.95}(r,6)$		.692	.868	

TABLE 39

ANALYSIS OF VARIANCE OF ACTUAL OXYGEN DEBT
FROM MINUTE 6 TO MINUTE 29
OF RECOVERY: GROUP C

Source of Variance	S.S.	df	M.S.	F.
Between people	2.075	3		
Within people	2.014	8		
treatment	.878	2	.439	2.323*
residual	1.136	6	.189	
total	4.089	11		

* $F_{.95}(2,6) < 5.14$

Actual Oxygen Debt (6 to 29) versus Lactic Acid Removal

The actual oxygen debt from minute six to minute twenty-nine versus the oxygen equivalents of the lactic acid removed during the associated period are presented in Table 40.

Correlated t-tests of the oxygen equivalent values of the lactic acid removed during recovery versus the corresponding oxygen debt (min. 6 to 29) revealed significant t-ratios between these values after the Group A, five minute run and after the Group B, fifteen minute run (Table 41). In spite of no significant differences found between the other pairs of measurements, it appeared that the oxygen equivalents of the lactic acid removed during this period consistently underestimated the actual lactic component during this period. The graph in Figure 6, which illustrates the underestimation of the lactic component by the oxygen equivalent values versus the duration of exercise, shows a tendency for the equivalent values to more closely approach the lactic component of the debt after the thirty minute runs, than after the five and fifteen minute runs for the Group A and Group B results. However, in the Group C data, the degree of underestimation by the lactic acid equivalents appeared to increase with increasing duration of exercise.

The graph in Figure 7 shows a lack of trend similar to Figure 6, when this underestimation was expressed as a percent duration from the lactic component and plotted versus the duration of exercise.

TABLE 40

LACTIC PORTION OF THE OXYGEN DEBT (MIN.6-29)
VERSUS OXYGEN EQUIVALENTS
OF LACTIC ACID REMOVED

Group A

	5 Minute	15 Minute	30 Minute
Lactic Component	.832	.783	.648
Oxygen Equivalent of Lactic Acid	.542	.362	.453

Group B

	5 Minute	15 Minute	30 Minute
Lactic Component	1.127	1.084	.745
Oxygen Equivalent of Lactic Acid	.453	.274	.583

Group C

	5 Minute	15 Minute	30 Minute
Lactic Component	.803	1.130	1.465
Oxygen Equivalent of Lactic Acid	.628	.908	.826

TABLE 41

CORRELATED t-TESTS OF THE OXYGEN EQUIVALENTS OF THE LACTIC ACID REMOVED DURING MINUTE 6 TO MINUTE 29 OF RECOVERY VERSUS THE CORRESOONDING ACTUAL OXYGEN DEBT

Group	5 Minutes	15 Minutes	30 Minutes
A	3.00*	2.69**	1.55**
B	.33**	3.87*	.59**
C	.51**	.52**	1.19**

*$t_{.95}$ (4) > 2.776

**$t_{.95}$ (4) < 2.776

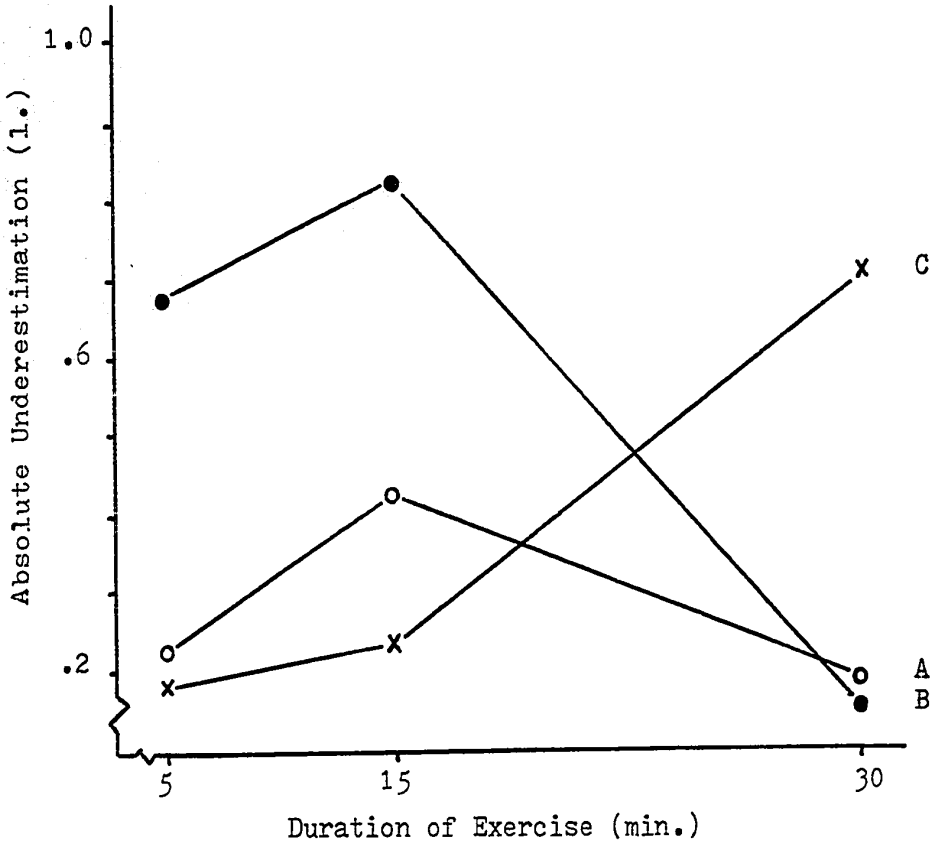


Figure 6:--Absolute Underestimation of Actual Oxygen Debt (min. 6 to 29) by Lactic Acid O2 Equivalents vs. Duration of Exercise

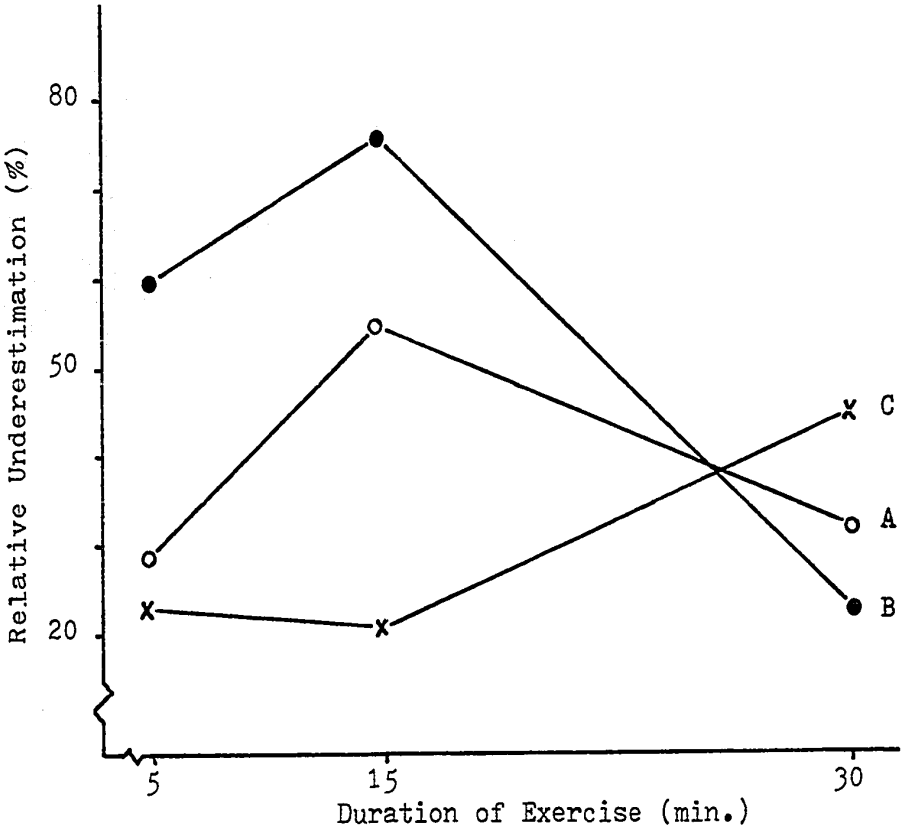


Figure 7:--Relative Underestimation of Actual Oxygen Debt (min. 6 to 29) by Lactic Acid O_2 Equivalents vs. Duration of Exercise

The Alactic Component

The alactic component measurements are presented in Table 42. Inspection of the graph in Figure 8 suggests an apparent consistent increase in the alactic component for all groups as a result of duration of exercise, up to the fifteen minute run. Between the five and fifteen minute runs, the Group A alactic component was increased by 33.3%, the Group B data, by 36.0%, and the Group C data, by 18.6%. The alactic components for Group A and Group B were increased a further 5.7% and 17.2%, respectively, when the duration of exercise was prolonged to thirty minutes. The Group C alactic component, however, decreased following the thirty minute run to a value which was 3.6% above that which had been present following the five minute run.

Special Conditions

The total oxygen and the oxygen debt from minute six to minute twenty-nine, as well as the oxygen equivalents of the lactic acid removed following the special condition runs, are presented in Table 43. The results of subject J.M. and subject A.K. showed a greater oxygen debt after running condition y* than condition x** (20.6% and 9.7%

*in special condition y, the subject ran for twenty minutes at his previously assigned intensity, with a two minute maximal bout of running presented at the beginning of the fifteenth minute of the run.

**in special condition x, the maximal bout of running was presented at the beginning of the fifth minute of the run.

TABLE 42

THE TOTAL OXYGEN DEBT AND THE ESTIMATED ALACTIC AND LACTIC COMPONENTS OBTAINED FROM THE THEORETICAL CURVES

Group A

Duration of run	Alactic Component	Lactic Component	Total
5 minutes	1.32	1.86	3.18
15	1.76	1.84	3.60
30	1.86	1.85	3.71

Group B

Duration of run	Alactic Component	Lactic Component	Total
5 minutes	1.11	3.04	4.19
15	1.51	2.38	3.89
30	1.77	2.32	4.09

Group C

Duration of run	Alactic Component	Lactic Component	Total
5 minutes	1.94	2.55	4.49
15	2.30	2.89	5.19
30	2.01	3.93	5.94

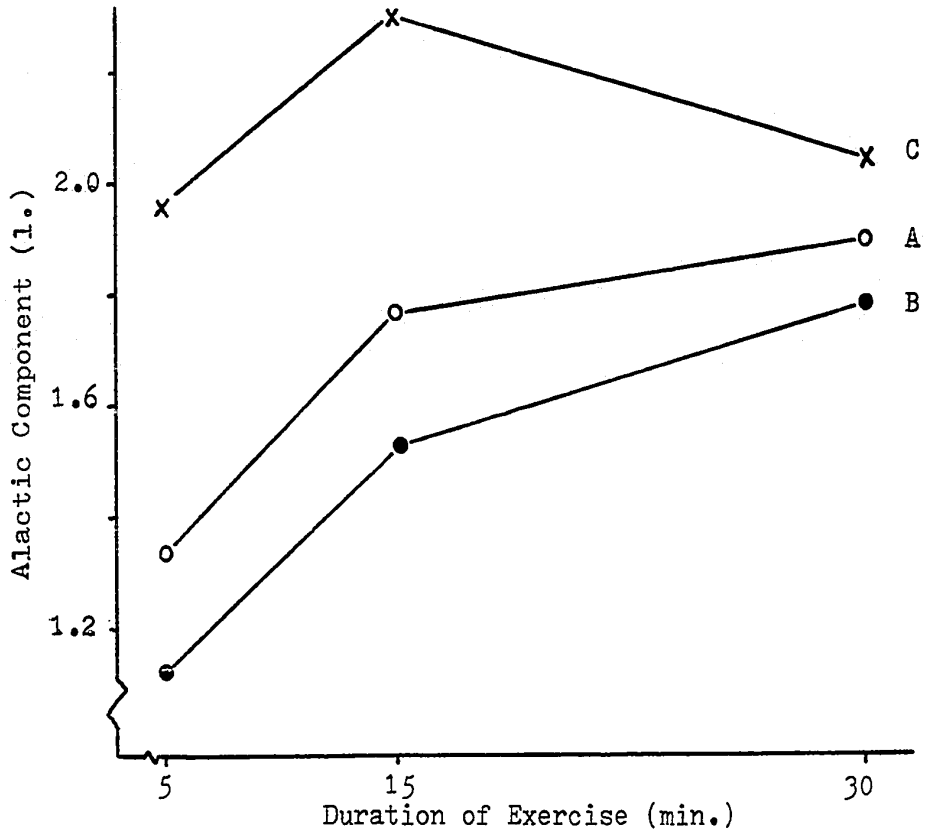


Figure 8:--Estimated Alactic Component vs. Duration of Exercise

TABLE 43

ACTUAL TOTAL OXYGEN DEBT, ACTUAL OXYGEN DEBT FROM MINUTE 6 TO 19, AND O_2 EQUIVALENTS OF LACTIC ACID REMOVED FOLLOWING SPECIAL CONDITIONS

Group A: Subj. A.K.

	Total O_2 Debt (l.)	O_2 Debt (l.) 6-19 min.	Lactic acid O_2 Equiv.
condition x	3.18	.61	1.49
condition y	3.49	.83	2.85

Group B: Subj. B.F.

	Total O_2 Debt (l.)	O_2 Debt (l.) 6-19 min.	Lactic acid O_2 Equiv.
condition x	4.02	1.03	.88
condition y	3.59	.95	1.75

Group C: Subj. J.M.

	Total O_2 Debt (l.)	O_2 Debt (l.) 6-19 min.	Lactic acid O_2 Equiv.
condition x	4.76	1.47	2.24
condition y	5.74	1.83	4.24

higher, respectively). The data for subject B.F., however, indicated a decreased total oxygen debt (by 10.7%) after running condition y. This data is illustrated in Figure 9.

Trends proportional to the one outlined above were found with the actual oxygen debt values from minute six to nineteen of recovery (Figure 10). The oxygen debt within this period was increased by 36.0% for subject A.K. and by 24.5% for subject J.M. as a result of condition y. The comparable measure for subject B.F. showed a decrease of 7.8% in the oxygen debt during this period.

The oxygen equivalent values for the lactic acid removed during this time, however, were, for all subjects, increased as a result of running condition y. For subject A.K., this increase was 91.3%; for subject B.F., 98.9%; and for subject J.M., 89.3% (Figure 11).

As opposed to the apparent consistent underestimation of the oxygen debt values by the equivalent data of the regular runs, there appeared to be, with one exception, a consistent overshooting of the oxygen debt data. The magnitude of this overestimation effect is illustrated in the graph in Figure 12. This tendency to overestimate appeared to be increased after the condition y runs as compared to the condition x runs, thus suggesting an increasing lack of a stoichiometric relationship as the presentation of the increased workload occurred closer to the end of the exercise period.

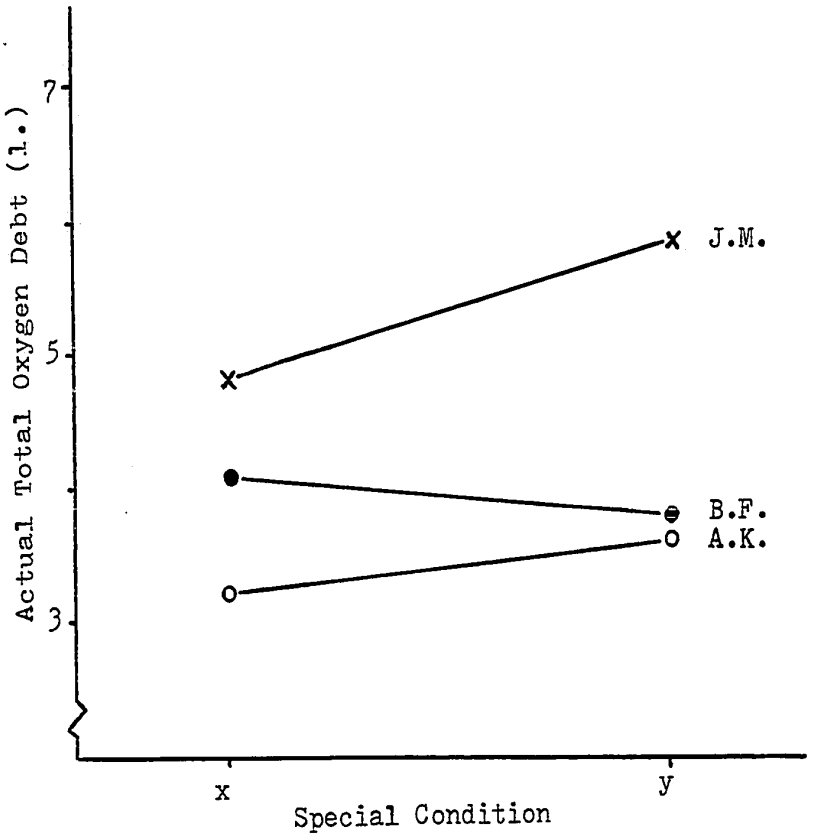


Figure 9:--Actual Total Oxygen Debt vs. Special Condition

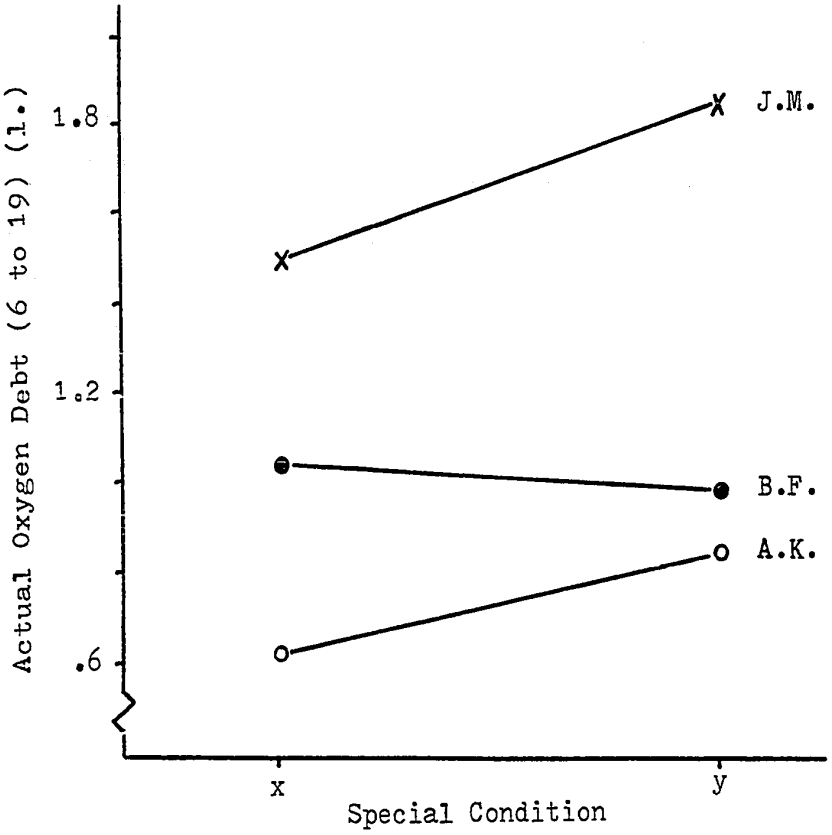


Figure 10:--Actual Oxygen Debt (min. 6 to 19) vs. Special Condition

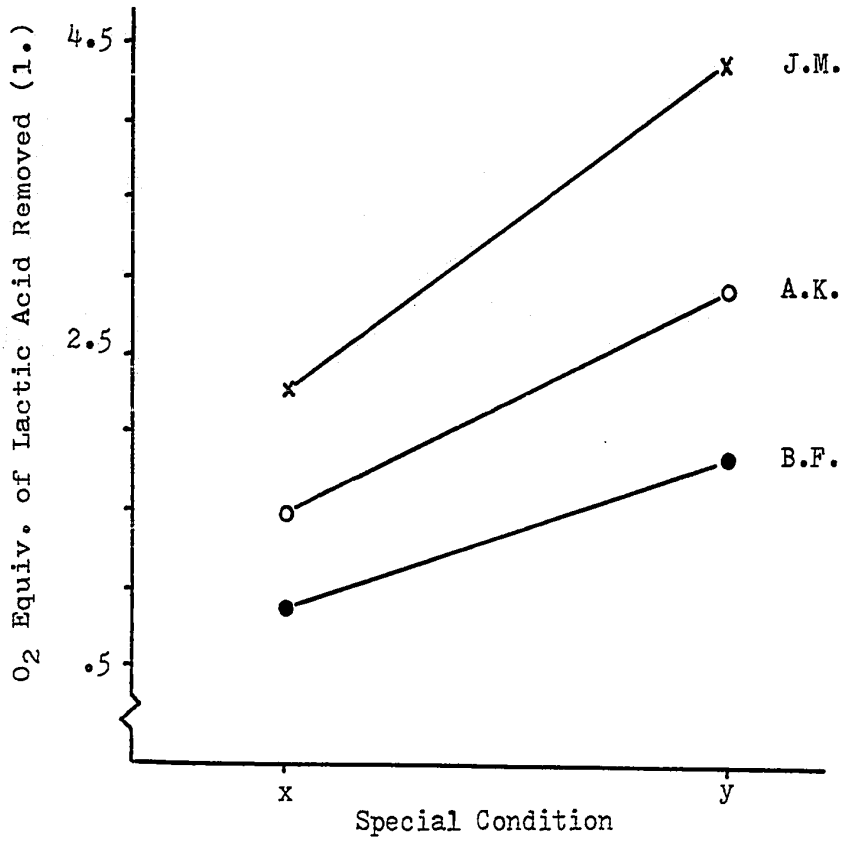


Figure 11:--O_2 Equivalents of Lactic Acid Removed (6 to 19) vs. Special Condition

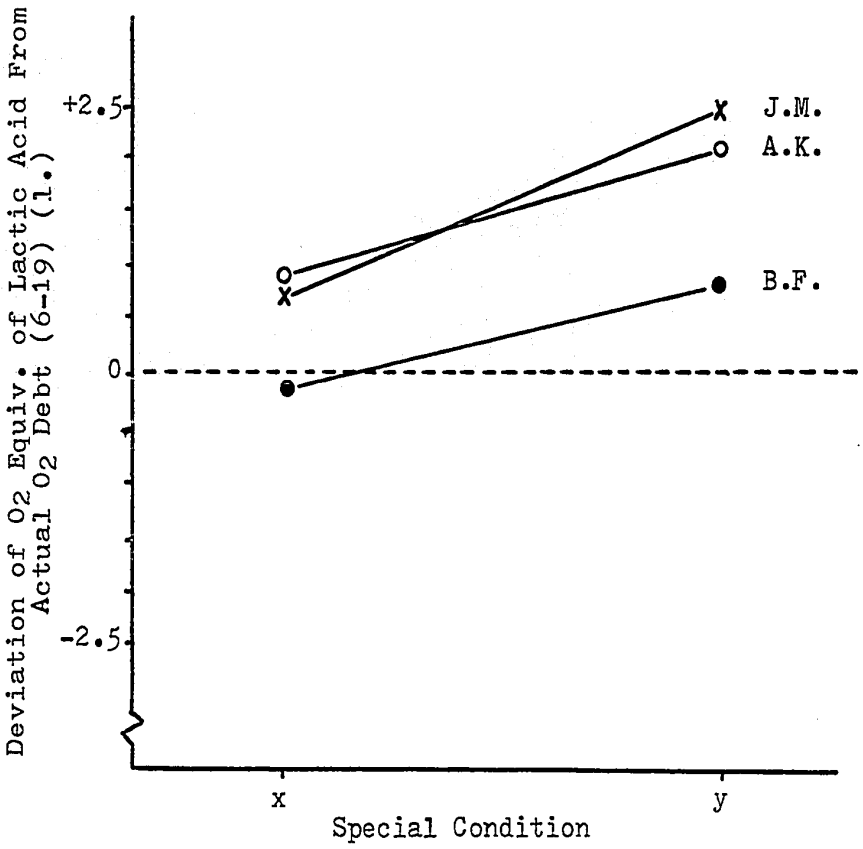


Figure 12:--Deviation of O_2 Equivalents of Lactic Acid Removed vs. Special Condition

CHAPTER V

DISCUSSION

Three groups of subjects, each group assigned a specific relative intensity of exercise, were tested at three durations of exercise to determine the effect of duration of exercise on total oxygen debt and the components of oxygen debt at each level of intensity. In addition, the relationship between the lactic acid removed, expressed in oxygen equivalents, and the lactic component of the oxygen debt repaid during the same time interval, were quantitatively compared to determine if a stoichiometric relationship existed after all durations and intensities of exercise. One subject from each intensity group performed two additional "special condition" runs to evaluate changes in the preceding parameters as affected by the time, during the steady state run, at which a short supramaximal bout of running was added.

Hypotheses were proposed which, if supported, would be in agreement with concepts of the Margaria theory of oxygen debt. In the event of rejection of these hypotheses at the .05 level of confidence, alternative hypothesis, which supported theories in conflict with that of Margaria, were accepted.

The Baseline for Oxygen Debt Determination

The determination of oxygen debt is based on the use of one of a number of frequently used baselines. Stainsby and Barclay (61), in their review of the subject of oxygen deficit and oxygen debt, illustrated the three baselines commonly used; pre-exercise, basal metabolic rate, and light exercise. Although they pointed out that the pre-exercise oxygen uptake "seems to yield reproducable results" they revealed the problems involved with the use of each baseline, and added that "one must raise the issues as to what recovery includes and what parts of it one wishes to study" (61).

In order to adequately test the Margaria et al. theory of oxygen debt, the point of asymptote of the oxygen consumption during the recovery period was utilized as the baseline. This use of the asymptote in lieu of the previously-used measures served a dual purpose: first, it allowed a more exact estimation of the true exponential nature of the lactic component of the oxygen debt; and, secondly, it was hoped that this procedure would parcel out a possible "third" component of the oxygen debt, which might be attributed to the more prolonged consequences of exercise. Margaria et al. (49) made reference to the possibility that a third component existed, but made no attempt at quantifying or explaining it. The research of Knuttgen (42) was a demonstration of how oxygen debt parameters could be affected by such "general body

disturbance" mechanisms as body temperature, electrolyte balance, hormone release, and sympathetic activity.

Therefore, it was felt that this use of a new base-line for the determination of oxygen debt provided a basis for the comparison of the lactic component and the lactic acid removed during the recovery period which has been unprecedented in previous research of this kind.

Exercise Oxygen Uptake

Analysis of the Group A oxygen uptake data revealed significant differences among the three durations of running for the measurements taken at the beginning of the fifth minute. Since this situation failed to occur at the higher intensities of running, it was assumed that this inconsistency of oxygen uptake at the same treadmill speeds was a result of a combination of two factors: (1) the subjects' awareness of the length of the run to be performed on the particular testing day; and (2) difficulty on the part of the subjects to adjust physiologically and psychologically to such a low speed of running. The latter point is further supported by the fact that, in many cases, the treadmill speeds for the subjects in Group A were sufficiently slow to allow fast walking instead of running, even though the subject were asked to run.

Similarly, this inability on the part of the subjects in Group A to be consistent in their response to the slow run may have carried over into recovery, resulting in different recovery patterns and thus causing lack of relationship

between the theoretical and actual oxygen debt parameters. Inspection of the theoretical curves for Group A revealed a wider scattering of the points after the sixth minute of recovery, thereby complicating attempts to estimate the lactic component. This, in turn, resulted in a significant difference between the two means of the oxygen debt from minute six to minute twenty-nine.

Total Oxygen Debt

It was assumed that the measurement of the total oxygen debt using the determined asymptote would result in lower oxygen debt values than those observed in previous literature in which exercise in a similar range of intensities was studied. This assumption was based on the observation that these previous studies has utilized baseline values which were considerably lower than the ones used in this investigation, thus including recovery oxygen in their oxygen debt measurement which was not included in the true lactic component. However, this was not the case. Knuttgen (42), using post-recovery oxygen uptake as his baseline, reported oxygen debt measurements of approximately 1.50 to 2.25 litres following exercise of 55% to 83% of maximum oxygen uptake. Similarly, Schneider *et al.* (59), also using post-exercise oxygen uptake as his particular baseline, observed oxygen debt levels of from 2.10 to 2.26 litres after exercise of 30% to 55% of maximum. These values appeared to be considerably less than the smallest oxygen debts of 3.33 to 3.57 litres found in the

Group A data. Due to the consideration that trained individuals are alleged to derive a higher percentage of the energy requirement of exercise from aerobic sources than untrained individuals (3) this discrepancy is unexplained.

The fact that the total oxygen debt measurements for all intensity groups failed to show significant increases as a result of duration of exercise has significant implications with respect to oxygen debt theory. Margaria et al. endorsed the concept of an exercise threshold intensity of two thirds of maximum oxygen uptake, above which total oxygen debt increased as time of exercise increased. The failure to find such a relationship in the Group C data warrants the rejection of the Margaria et al. theory concerning this particular concept.

Schneider et al. (59) from the results of their study of oxygen debt following exercise of from thirty to fifty-five percent of maximum oxygen uptake, concluded that the oxygen consumption during exercise within this range of intensity was a reflection of tissue oxygen need, and that any debt incurred at the beginning of exercise was neither repaid nor accumulated during the exercise. However, no claims that this relationship existed above this intensity were made. From the results of this study, it is postulated that a similar relationship may exist at levels of exercise above Margaria's threshold theory.

In doing this, the contentions of Stainsby and Barclay (61) and Saiki (57), and Gisolfi et al. (20) that oxygen

consumption during exercise included metabolic turnover, are not necessarily refuted. Metabolite turnover, such as lactic acid oxidation, could occur during exercise, with no subsequent effects on the total oxygen debt, if one component were transformed into another. The possibility was expressed by Gisolfi et al. (20) and inferred by Saiki (57), that lactic component might be transformed into alactic component during prolonged steady state running. In addition, Margaria (46) cited research indicating that perhaps the alactic component was transformed into lactic component during, and immediately following, steady state or strenuous exercise. These trends as they relate to the data of the present study are examined later in this chapter. Nonetheless, the fact remains that the threshold concept with respect to total oxygen debt as originally proposed by Margaria, was rejected by the results of this study.

Lactic Component

Knuttgen (42) observed that his estimated lactic component increased, as a function of duration of exercise at 55% to 83% of maximum oxygen uptake, from approximately 1.15 to 1.50 litres. Although the levels observed in this study appeared to exist in lower quantities than Knuttgen's lower range (.648 was the lowest measured lactic component in this study) the upper ranges were comparable (1.465 in this study). This suggests that the asymptote may not have excluded as much confounding recovery oxygen as was originally intended, but that Knuttgen's lactic component

increases were due to the characteristics of his subjects.

The significant decrease in the lactic component (oxygen debt from minute six to twenty-nine of recovery) as a result of duration of exercise in the Group B data, supported the previous disagreement with the Margaria et al. threshold concept. Since the total oxygen debt values remained unchanged irrespective of duration of exercise at all intensity levels, it was assumed that a gradual transition of lactic component into alactic component occurred, as contended by Gisolfi et al. (20) and Saiki (57). This assumption was enhanced by an apparent, although not statistically significant, increase in the alactic component for this group. The opposite results were reported by Knuttgen (42) at sixty percent of maximum oxygen uptake. In view of this relationship, the concept of the oxygen consumption during exercise as a reflection of the need for oxygen by the working tissues, as well as metabolic turnover, would necessarily assume the oxidation of lactic for the purposes of resynthesis of high energy phosphates and other alactic mechanisms. However, since research in this aspect has been lacking, this seeming relationship necessarily remained unexplained.

Alactic Component

The alactic components of up to 2.3 litres following the Group C thirty minute run were considerably higher than the 1.5 litre alactic component reported by Knuttgen, and

were also higher than those reported by Henry and DeMoor (27). However, the alactic components, as estimated by inspection in the present study, were considered accurate in view of the curve validation procedures conducted.

Henry (25) reported that the magnitude of the alactic component was dependent on the level of oxygen consumption at time zero of recovery, and that the velocity constant (k_1) for this component was fairly constant. This was not found to be the case in the results of the study. The alactic component appeared to increase with increasing duration of exercise, with one exception. Since the oxygen consumption at time zero of recovery for all runs of each group were statistically the same, it was primarily variation in the velocity constant that resulted in the increased component.

These tendencies for the alactic component to increase with increasing duration of exercise suggested a possible "general body disturbance" (42) factor confounding this component. Since the primary metabolite of alactic debt, creatine phosphate, has been shown by Karlsson et al. (35) to be depleted to the same extent after two to three minutes irrespective of intensity of exercise, the possibility exists that such factors as body temperature and ionic imbalance may not have been parcelled out by using the asymptote alone. These factors may conceivably have played an increasing role in the alactic component as a function of time. In addition, the possibility that lactic component was transformed into alactic component, as

postulated by Gisolfi et al. (20) and Saiki (57), exists. This relationship was expressed by Rowell (56) who, due to the stability of the total debt that he observed in spite of decreased lactic acid concentration after longer runs, proposed that blood lactic acid concentration time dependent while oxygen debt is not. However, in light of the fact that the alactic component data was not subjected to statistical analysis, these suggestions are based on conjecture alone.

The Relationship of Lactic Acid Removal vs. Oxygen Debt

In order to related lactic acid removal and oxygen debt parameters, the stoichiometric relationship for the oxidation of lactic acid ($2CH_3CHOHCOOH + O_2 = 2CH_3COCOOH + 2H_2O$) was used to transform the lactic acid data into units capable of comparison with the recovery oxygen data. According to this relationship, one mole of oxygen (22.4 litres) is required to remove two moles (180 grams) of lactate (29).

Stainsby and Barclay (61) pointed out that lactate can be taken up and used as a substrate source and oxidized, thus not contributing to recovery oxygen. Similarly, Rowell (56) stated that the relationship of oxygen consumption during recovery and lactic acid removal was affected by the capacity of the resting tissues to substitute lactate for other substrates. Barnard, Foss and Tipton (70), in their research involving the role of the

Cori cycle in oxygen debt repayment, suggested that the oxidation of lactate to pyruvate did not contribute to oxygen debt by stating:

> the involvement of lactate in O_2 debt has been eliminated when conversion of lactate back to glucose is blocked (7)

Yet, Boxer (8), described lactic acid as one of the rare dead ends in a mammalian metabolic path, and suggested:

> Since it is not directly oxidized by the mitochondria, and not aminated or decarboxylated, the only way for the three carbons to return to the main stream of metabolic events is by reoxidation to pyruvic acid by way of DPN-dependent lactic dehydrogenase system (8).

Similarly, Wasserman (69) points out that the lactate molecule, regardless of whether it is excess or not excess and regardless of its ratio to pyruvate, requiring one-half molecule of oxygen or equivalent oxidation by another source before it can be converted to pyruvate.

Therefore, the suggestions cited above, were considered ample justification for the use of a stoichiometric equation for the determination of the oxygen equivalents necessary for the removal of lactic acid.

In relation to the results found in the present study, the lack of significant differences (except in two cases) between lactic component measurements and the corresponding oxygen equivalent values of the lactic acid removed suggests

an acceptance of the Margaria et al. concept of concerning this relationship. A consistent underestimation of the oxygen debt by the lactic acid equivalents, although not statistically significant was similar to an "oxygen wasting" effect reported by Barnard, Foss and Tipton (7), who reported that the increase in oxygen consumption induced by lactate infusion was in excess of that required for its oxidation. Similar results were reported by Levy (44), who was unable to account for the effect.

Previous research, in attempting to explain this apparent underestimation effect, has relied on explanations that infer the disappearance of lactate without corresponding oxygen utilization. It is proposed that, since lactic acid oxidation ultimately must involve molecular oxygen, any deviation in the lactic acid removed and the oxygen is due to a time lag effect. This concept, as outlined by Kayne and Alpert (39) and Boxer and Devlin (8) is considered in more detail below.

Special Conditions

The increase in total oxygen debt and lactic component oxygen debt values for subject J.M. and A.K., as a result of condition y, suggested a possible repayment of the oxygen debt when the length of time at steady state exercise following the maximum work bout was increased. This relationship seemed in agreement with the concept of Gisolfi et al. (20) who suggested a gradual repayment of oxygen debt when a maximal bout of exercise was followed by a period steady

state exercise. However, their conclusions were based entirely on their lactic acid data. On the other hand, Schneider et al. (59) postulated that, when an increase in intensity of exercise occurred during a submaximal run, the debt resulting from that increase alone would be repaid when the steady state was resumed. These relationships were not substantiated by the results observed in this study.

However, the consistent increase in the oxygen equivalents of the lactic acid removed and the seeming increase in the deviation between the two parameters for condition y as opposed to condition x suggested a departure from the stoichiometric relationship found between oxygen debt and oxygen equivalent values following the regular runs. Kayne and Alpert (39) hypothesized that the consumption of oxygen was directly related to the number of electrons transferred from substrate to molecular oxygen in the mitochondria. According to their concept, since $NADH_2$ may be impermeable to the mitochondrial membrane, a piling up of electrons outside the mitochondria might occur, thus resulting in increased lactate levels. However, the rate of oxygen consumption would be limited by the rate of transfer of electrons across the mitochondria membrane, and not be lactic acid concentration.

Boxer and Devlin (8) cite research demonstrating that carefully isolated "intact" rat liver mitochondria do not oxidize added $NADH_2$ and thus extramitochondrial $NADH_2$ is

presumably not a substrate for oxidation by way of the electron transport system. NAD is said to act merely as a shuttle system to redistribute extramitochondrial electrons or hydrogen ions and allow glycolysis to proceed anaerobically (39).

It is therefore hypothesized that, during the steady state runs, the exchange of hydrogen ions from reduced NAD into the mitochondria did not result in an accumulation of $NADH_2$ outside the mitochondria to an extent that would show significant differences between oxygen consumption and lactic acid removal: in fact, the alleged "shuttle systems" were able to keep pace with the oxidation of lactic acid. In the case of the special conditions, it is hypothesized that the added increase in workload resulted in a temporary piling up of hydrogen ions outside the mitochondrial membrane, which then tended toward equilibrium as the steady state was resumed. According to this concept, the overestimation of the lactic component by the lactic acid concentration would depend on the period of time between the increased work bout and the end of exercise. The results of this investigation suggest this as a possible mechanism.

The Hypotheses

In view of the results observed, the following conclusions, with respect to the hypotheses originally drawn, are justified.

1. Hypothesis #1 is accepted since no data were found to refute the Margaria *et al.* theory that total oxygen debt does not change as a result of duration of exercise below the two-thirds of maximum threshold.

2. Hypothesis #2 is rejected, thus rejecting the Margaria *et al.* theory that lactic component does not change as a result of duration of exercise below the two-thirds of maximum threshold. In its stead, the alternative hypothesis H_0: $b_1 \neq b_2 \neq b_3$ is accepted. In addition, it is proposed that the lactic component of the oxygen debt decreases with increasing duration of exercise of approximately sixty five percent of maximum oxygen uptake.

3. Hypothesis #3 is rejected, thus rejecting the Margaria *et al.* theory that total oxygen debt increases with duration of exercise above the two-thirds of maximum threshold level. In its stead, the alternative hypothesis H_0: $C_1 = C_2 = C_3$ is accepted.

4. Hypothesis #4 is rejected, thus rejecting the Margaria *et al.* theory that the lactic component of the oxygen debt increases with increasing duration of exercise above the two-thirds of maximum threshold level. In its stead, alternative hypothesis H_0: $d_1 = d_2 = d_3$ is accepted.

5. Hypothesis #5 is accepted since no evidence was found to reject the Margaria *et al.* theory that the lactic component and the lactic acid removal during recovery are stoichiometrically related. However, it is proposed that this theory is tenable, in light of the results observed following the two special condition runs.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The total oxygen debt, the components of the oxygen debt, and the relationship between the lactic component and the lactic acid removed during recovery were measured at three intensities of submaximal exercise to test the Margaria *et al.* (49) theory of oxygen debt. The recovery oxygen consumption curve asymptote was used as a baseline in an attempt to eliminate "body disturbance" (42) factors which have affected the results of similar research in the past. In addition, three subjects each performed two special condition runs at one of the three intensities in an attempt to clarify some of the relationships observed in the regular runs.

The results indicated: (1) total oxygen debt is not affected by duration of exercise at approximately 50%, 65% and 80% of maximum oxygen uptake, thus refuting the Marbaria *et al.* threshold concept; (2) that the lactic component decreases as the time of exercise at 65% of maximum oxygen uptake increased, thus refuting the Margaria *et al.* theory that this component does not change as a result of time at this intensity; (3) that the lactic component does not increase as a result of increasing the

duration of time at 80% of maximum oxygen uptake, thus refuting the Margaria et al. theory that proposes the contrary; and (4) that a stoichiometric relationship exists between the lactic component of the oxygen debt and the lactic acid removed following submaximal exercise. However, this latter concept appears to be tenable when the submaximal exercise is interrupted by a bout of supramaximal. work.

General Conclusions

In light of the results observed in this study, it appears justifiable to suggest a mechanism more directly related to the oxygen debt phenomenon than the removal of lactic acid. The work of Kayne and Alpert (39) and Boxer and Devlin (8) emphasize the possibility that the permeability of the mitochondrial membrane to intermediate hydrogen-carrying compounds, and the availability of alternate shuttle systems to carry hydrogen electrons from reduced $NADH_2$ outside the membrane to oxidized NAD inside, may be the actual regulators of oxygen consumption during recovery. In terms of this relationship, lactic acid accumulation outside the mitochondria is probably irrelevant, although a coincidental relationship may exist. Therefore, it is hypothesized that the answer to the oxygen debt mechanism following maximal as well as submaximal exercise will be found by means of study of the mitochondrial NAD - cytoplasm NAD transfer mechanisms, since this equilibrium seems to be the rate limiting factor in oxygen uptake during recovery.

The Margaria *et al.* theory of oxygen debt and the concomitant removal of blood lactic acid during recovery from mild and severe exercise has never been tested directly, in spite of published reports indicating that this theory may contain misconceptions. It appears that the primary obstacles to the attempts at validation or refutation of the theory have been (1) the number of baselines available for the determination of oxygen debt, (2) the lack of a sound rationale accompanying the use of any particualr baseline, (3) confusion with respect to the fate of lactic acid as it relates to oxygen debt, and (4) the lack of attempts to enumerate the components of the oxygen debt and explain them in terms of their biochemical and physiological determinants.

The rejection of the Margaria *et al.* theory regarding the threshold concept was the verification of the rejection merely implied in previous research attempts. Similarly, the observation that the lactic component of the oxygen debt was decreased with increasing duration of exercise at sub-threshold intensities added weight to similar assumptions from past research based on total oxygen debt and lactic acid data.

The apparent difference in the relationship between lactic acid removal and oxygen debt repayment between steady state exercise and exercise of varied intensities holds important implications for the widely-accepted lactic acid-oxygen debt phenomenon. Perhaps the answer to the question regarding whether or not lactic acid is the

"primary fatigue substance" cannot be answered by studying steady state exercise, after which the lactic acid removal and oxygen debt repayment may be "coincidental, not causal" (39).

It must be kept in mind that this study was conducted on the basis of specific assumptions regarding the oxygen debt curve. Although the curve contains two well-defined exponential functions, the assignment of each to a specifically defined set of biochemical and physiological events is probably naive at best. In addition, the "parcelling out" of an unwanted portion of the oxygen debt curve is necessarily based on conjecture.

Recommendations for Further Research

It is apparent from review of the literature that the concept of oxygen debt is not as simple as originally proposed. The following set of recommendations is presented in the hope that future research conducted along these lines will ultimately unravel the mechanisms, and their interactions, between the various compartments of oxygen debt.

1. This research was conducted on the assumption that the oxygen debt curve consists of two primary components, involving lactic and alactic mechanisms, and a possible third component, related to the more prolonged consequences of muscular exertion. The possibility exists that there are more than two primary components. Similarly, the

separation of the curve into its distinct components must be further examined in terms of the biochemical and physiological determinants of each.

2. The use of the proper baseline in the determination of oxygen debt shall continue to plague physiologists until a scientifically sound rationale determines the proper value to utilize.

3. Animal research involving the investigation of mitochondrial membrane permeability, how it is affected by rate of metabolism and accumulation of substrate, and how it affects the relationship between lactic acid and oxygen will lend clues to the oxygen debt mechanism.

4. Investigation into the area of the direct causes of muscular fatigue, still unexplained, will undoubtedly link present thought with theory of the past.

APPENDIX A

TABLE 44

INDIVIDUAL LACTIC ACID DATA
GROUP A

Five Minute Run

Time	Lactic acid concentration (mg%)			
	B.G.	P.M.	A.K.	$\bar{X}$
Pre-exercise	5.35	12.20	8.50	8.68
3 minutes	12.30	14.15	20.92	15.79
6	17.60	10.80	20.50	16.30
9	9.90	14.35	22.38	15.54
19	15.00	6.92	13.21	11.71
29	9.92	4.51	13.38	9.27

Fifteen Minute Run

Pre-exercise	2.50	15.90	11.55	9.98
3 minutes	12.40	17.75	24.80	18.32
6	16.60	19.85	23.38	19.94
9	12.40	20.70	23.20	18.77
19	4.88	10.98	23.80	13.22
29	11.70	15.35	12.75	13.27

Thirty Minute Run

Pre-exercise	16.80	14.68	21.05	17.51
3 minutes	15.95	11.70	31.35	19.67
6	28.50	12.28	23.86	21.55
9	28.43	9.00	18.82	18.75
19	18.58	9.70	17.10	15.13
29	29.66	8.78	7.37	15.27

TABLE 45

INDIVIDUAL LACTIC ACID DATA
GROUP B

Five Minute Run

Time	Lactic acid concentration (mg%)				
	J.S.	D.B.	B.F.	G.P.	$\bar{X}$
Pre-exercise	6.94	18.60	13.90	11.97	12.85
3 minutes	3.70	28.90	11.18	7.20	12.25
6	5.65	23.70	15.20	8.15	13.18
9	1.48	21.50	10.40	8.50	10.47
19	7.25	12.25	6.27	9.55	8.83
29	3.95	11.98	4.55	7.50	7.00

Fifteen Minute Run

Pre-exercise	16.70	8.30	12.30	8.80	11.53
3 minutes	17.85	24.30	17.80	19.15	19.78
6	13.85	19.70	16.68	19.60	17.46
9	17.70	25.40	11.15	19.60	18.46
19	15.55	11.60	6.08	10.00	10.81
29	14.50	18.60	4.80	16.95	13.71

Thirty Minute Run

Pre-exercise	10.75	10.70	18.65	8.66	12.19
3 minutes	11.75	37.50	26.80	32.70	27.19
6	9.95	28.30	29.30	39.65	26.80
9	1.50	32.77	23.38	32.30	22.49
19	1.65	22.25	26.25	7.95	14.53
29	3.15	21.35	31.70	21.82	19.51

TABLE 46

INDIVIDUAL LACTIC ACID DATA
GROUP C

Five Minute Run

Time	Lactic acid concentration (mg%)				
	G.M.	H.B.	J.M.	K.C.	$\bar{X}$
Pre-exercise	12.00	6.55	7.65	16.15	10.59
3 minutes	20.30	22.20	23.45	43.55	27.38
6	14.60	21.60	28.60	26.20	22.75
9	31.45	10.25	20.70	31.30	23.43
19	11.15	10.70	8.30	38.55	17.18
29	9.00	6.85	7.95	33.39	14.30

Fifteen Minute Run

Pre-exercise	14.80	6.38	12.00	2.40	8.90
3 minutes	31.45	26.16	28.47	38.75	31.21
6	25.10	23.34	41.45	23.80	28.43
9	24.70	21.22	41.60	12.20	24.93
19	15.90	9.75	18.78	12.10	14.13
29	13.35	5.80	13.67	9.55	10.59

Thirty Minute Run

Pre-exercise	9.70	9.80	12.60	7.65	9.94
3 minutes	30.45	16.48	60.50	31.85	34.82
6	23.25	16.25	52.25	25.83	29.40
9	20.70	27.35	45.35	22.65	29.01
19	25.60	18.48	27.75	7.20	19.76
29	13.80	12.33	33.90	7.28	16.83

TABLE 47

LACTIC ACID DATA FOR SPECIAL CONDITIONS

Group A: Subject A.K.

Time	Concentration mg%		O_2 equivalent of lactate removed	
	Cond. X	Cond. Y	Cond. X	Cond. Y
Pre-ex	15.95	7.40		
3 min.	43.25	88.10		
6 min.	31.75	92.70	1.49	2.85
9 min.	39.00	65.50		
19 min.	20.90	45.50		

Group B: Subject B.F.

Time	Concentration mg%		O_2 equivalent of lactate removed	
	Cond. X	Cond. Y	Cond. X	Cond. Y
Pre-ex	25.00	12.20		
3 min.	37.00	64.45		
6 min.	36.40	53.80	.877	1.75
9 min.	34.20	48.45		
19 min.	24.60	39.80		

Group C: Subject J.M.

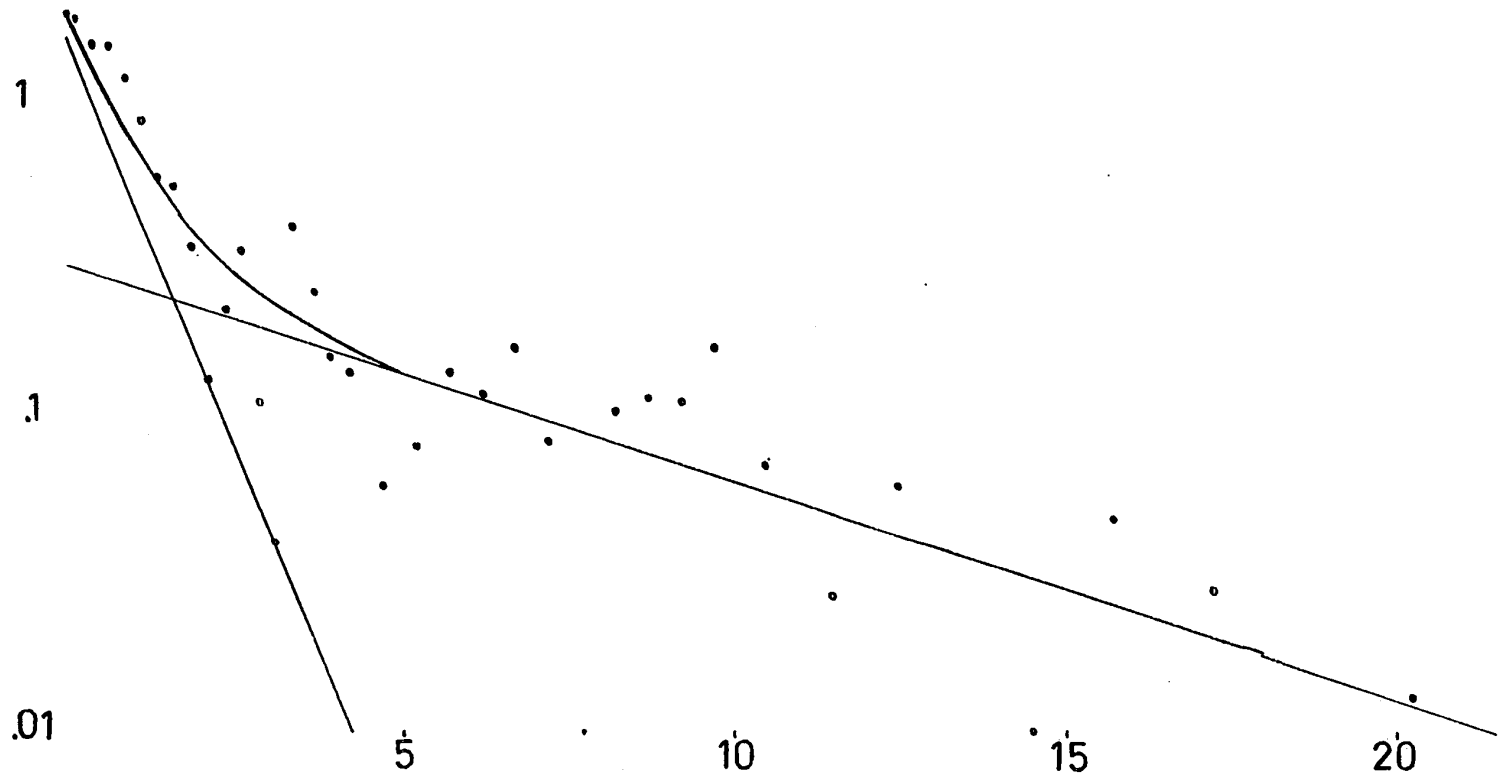
Time	Concentration mg%		O_2 equivalent of lactate removed	
	Cond. X	Cond. Y	Cond. X	Cond. Y
Pre-ex	20.35	6.70		
3 min.	57.85	113.50		
6 min.	47.30	85.40	2.24	4.24
9 min.	42.30	80.00		
19 min.	21.50	44.75		

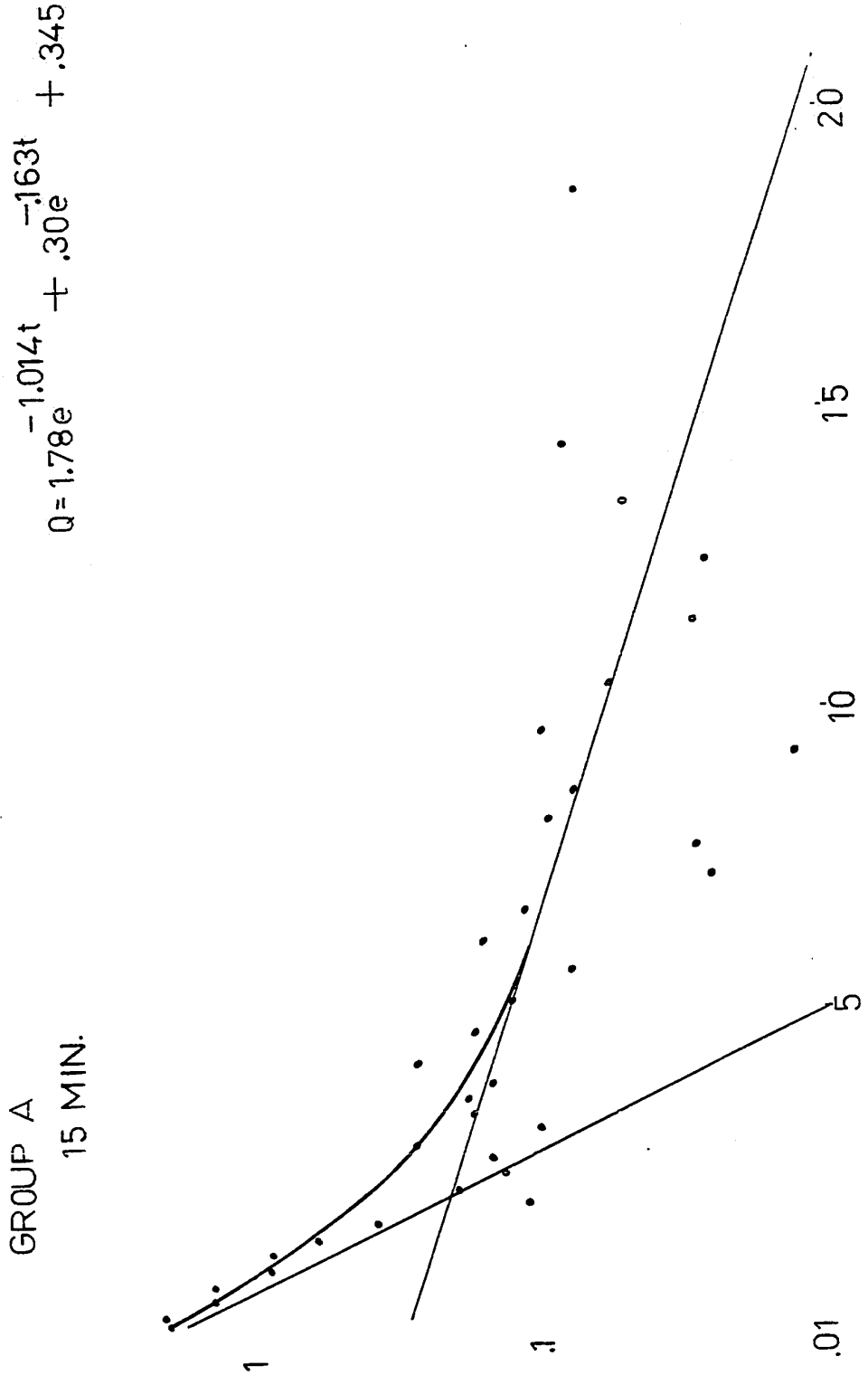
APPENDIX B

GROUP A

5 MIN.

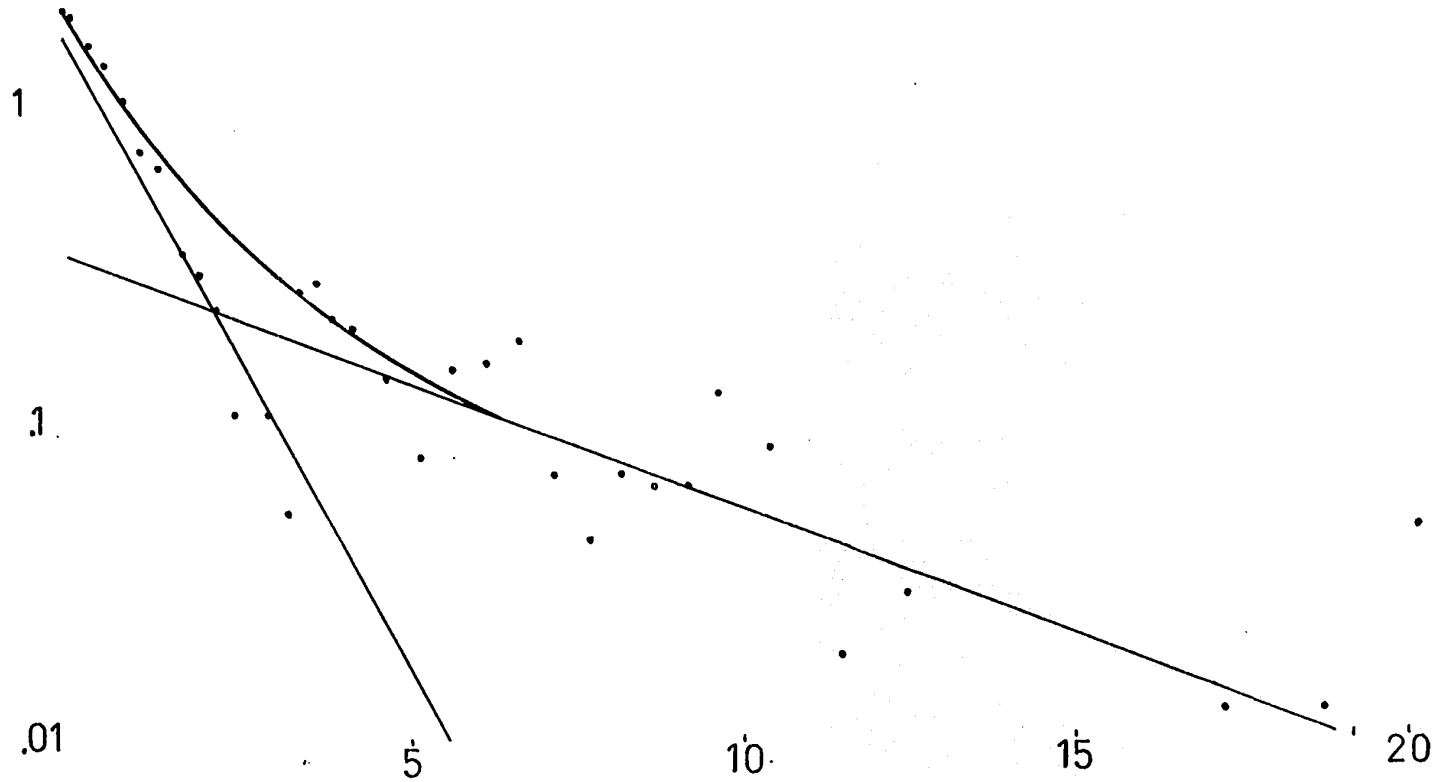
$Q = 1.57e^{-1.188t} + .30e^{-.161t} + .340$




GROUP A
15 MIN.
$Q = 1.78e^{-1.014t} + .30e^{-.163t} + .345$
1
.1
.01
5
10
15
20

GROUP A
30 MIN.

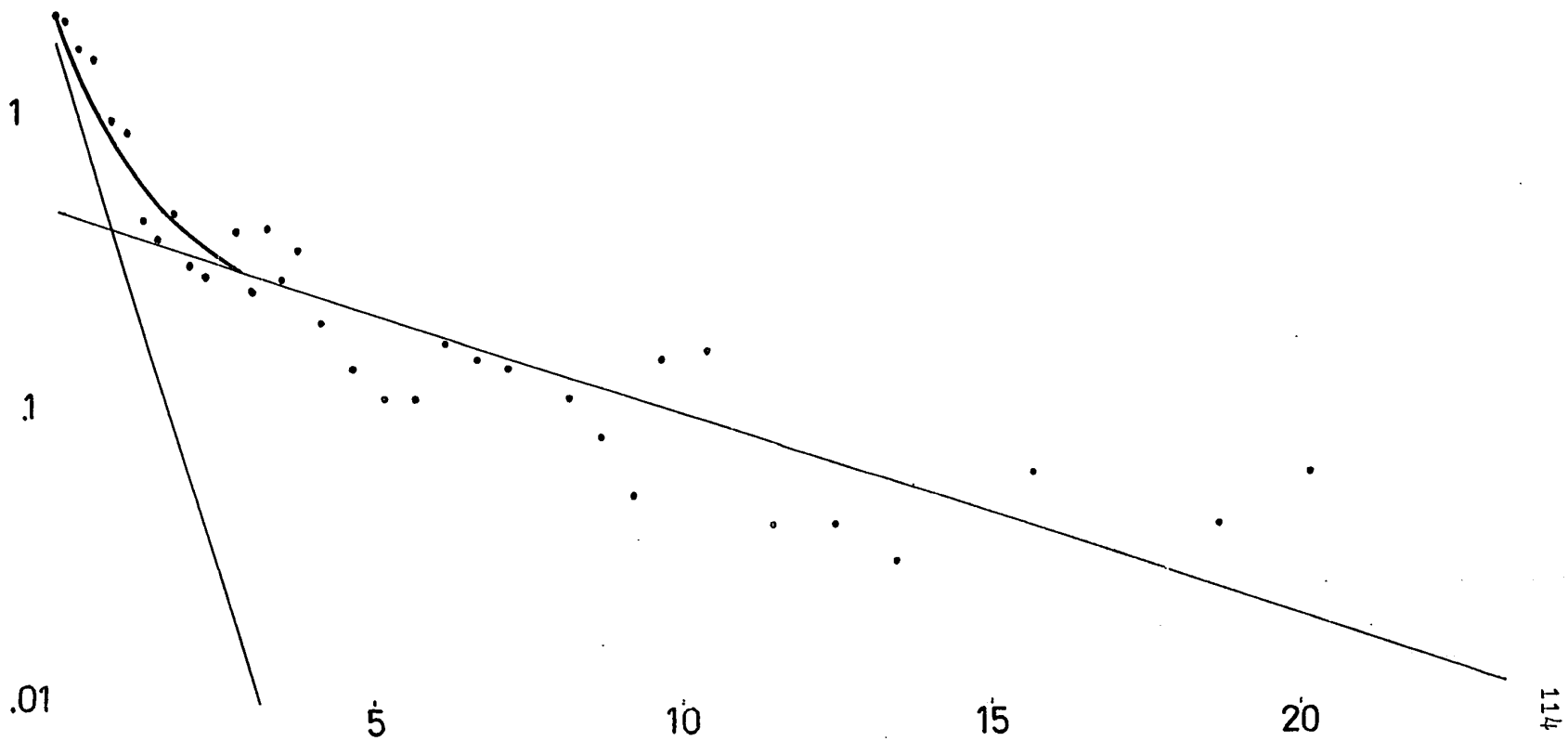
$Q = 1.68e^{-.903t} + .34e^{-.184t} + .375$

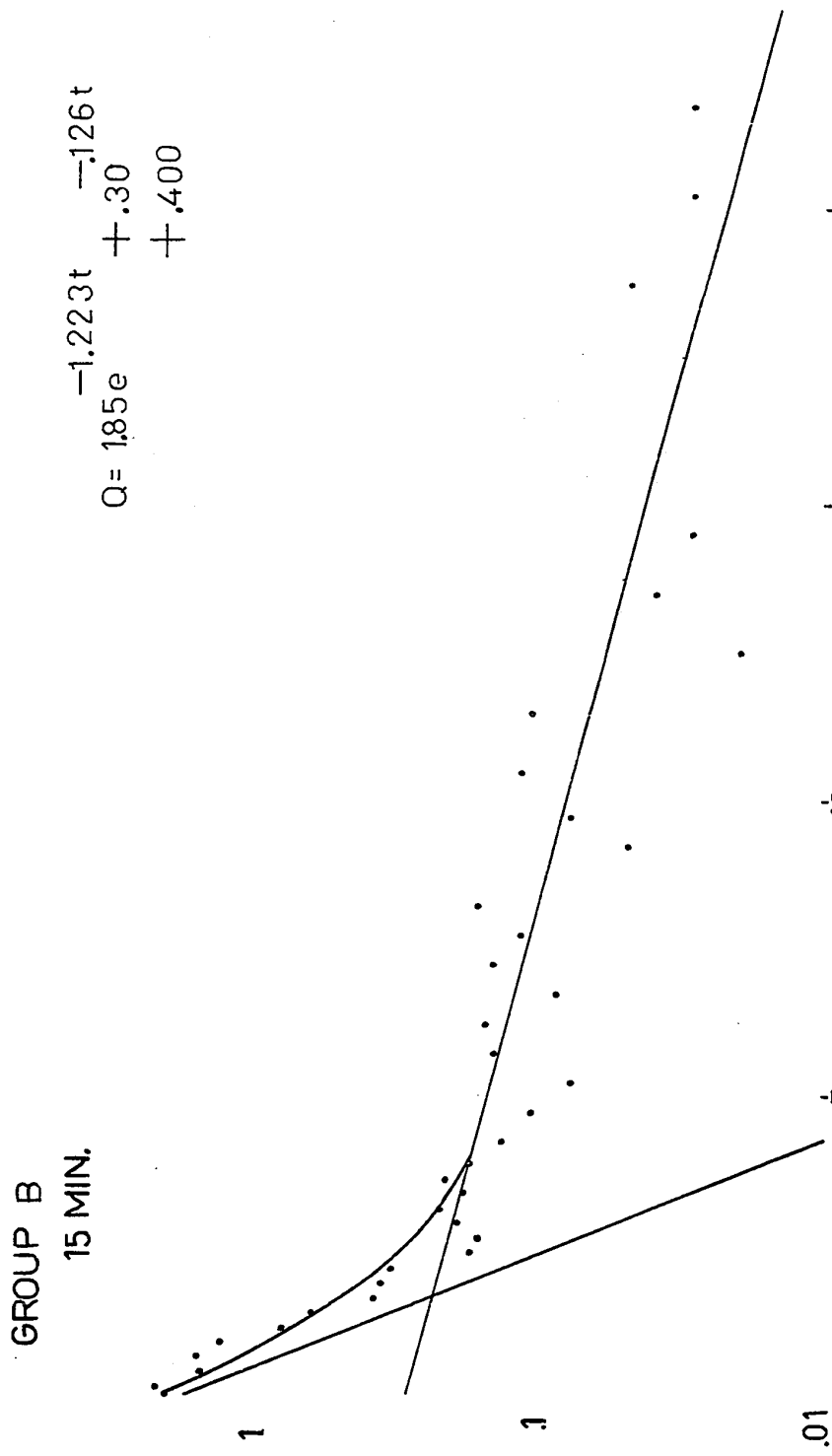


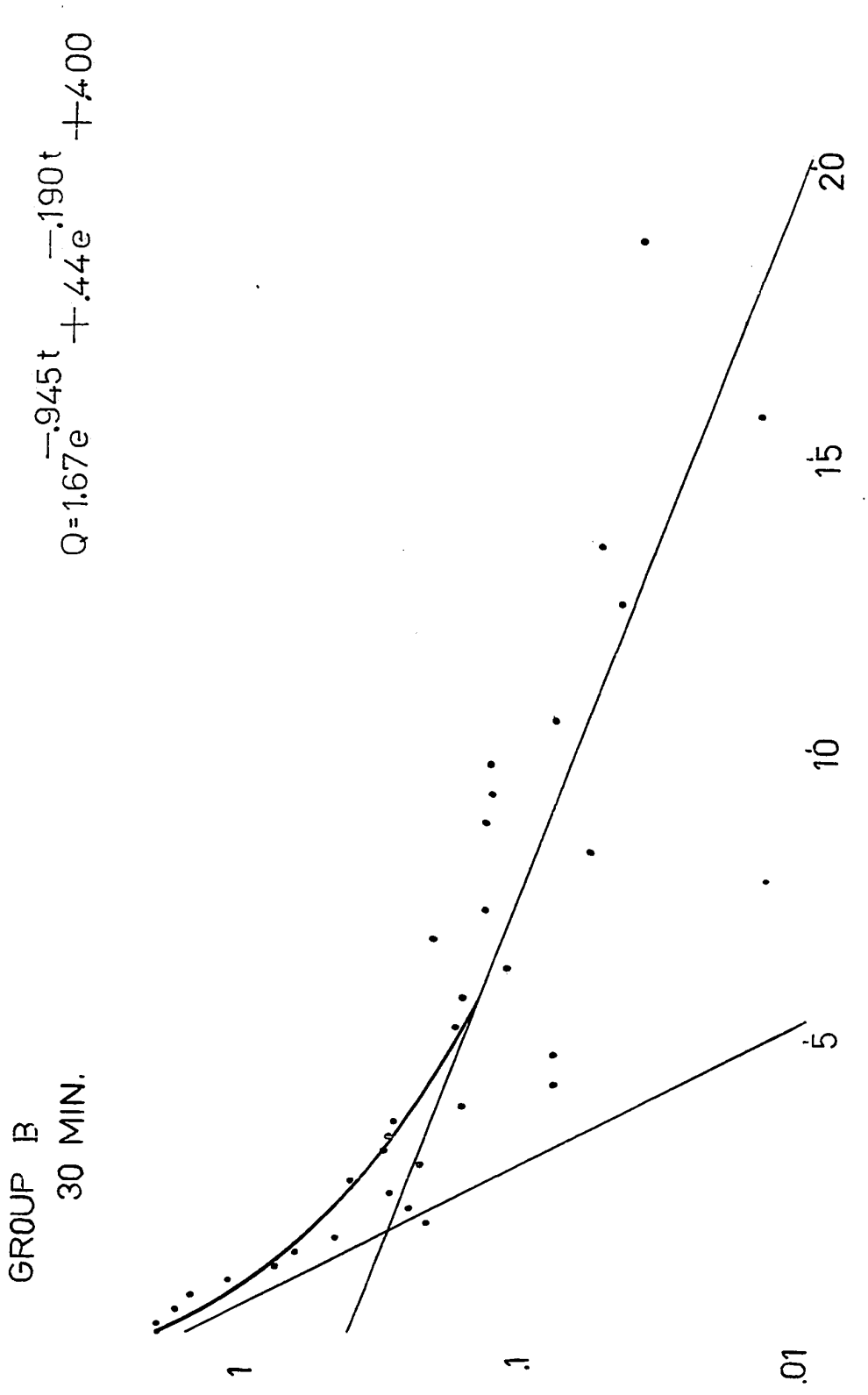
GROUPB

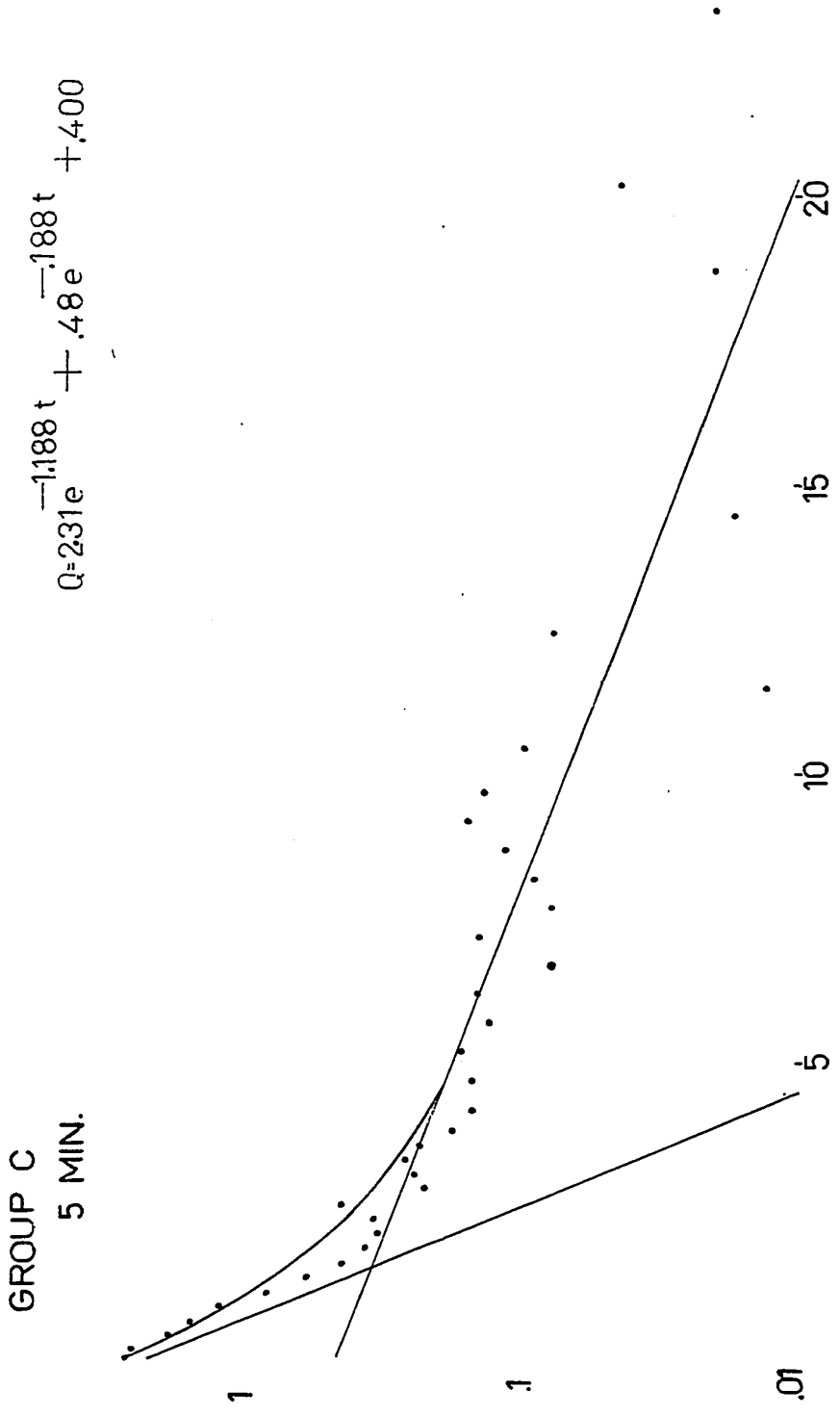
5 MIN.

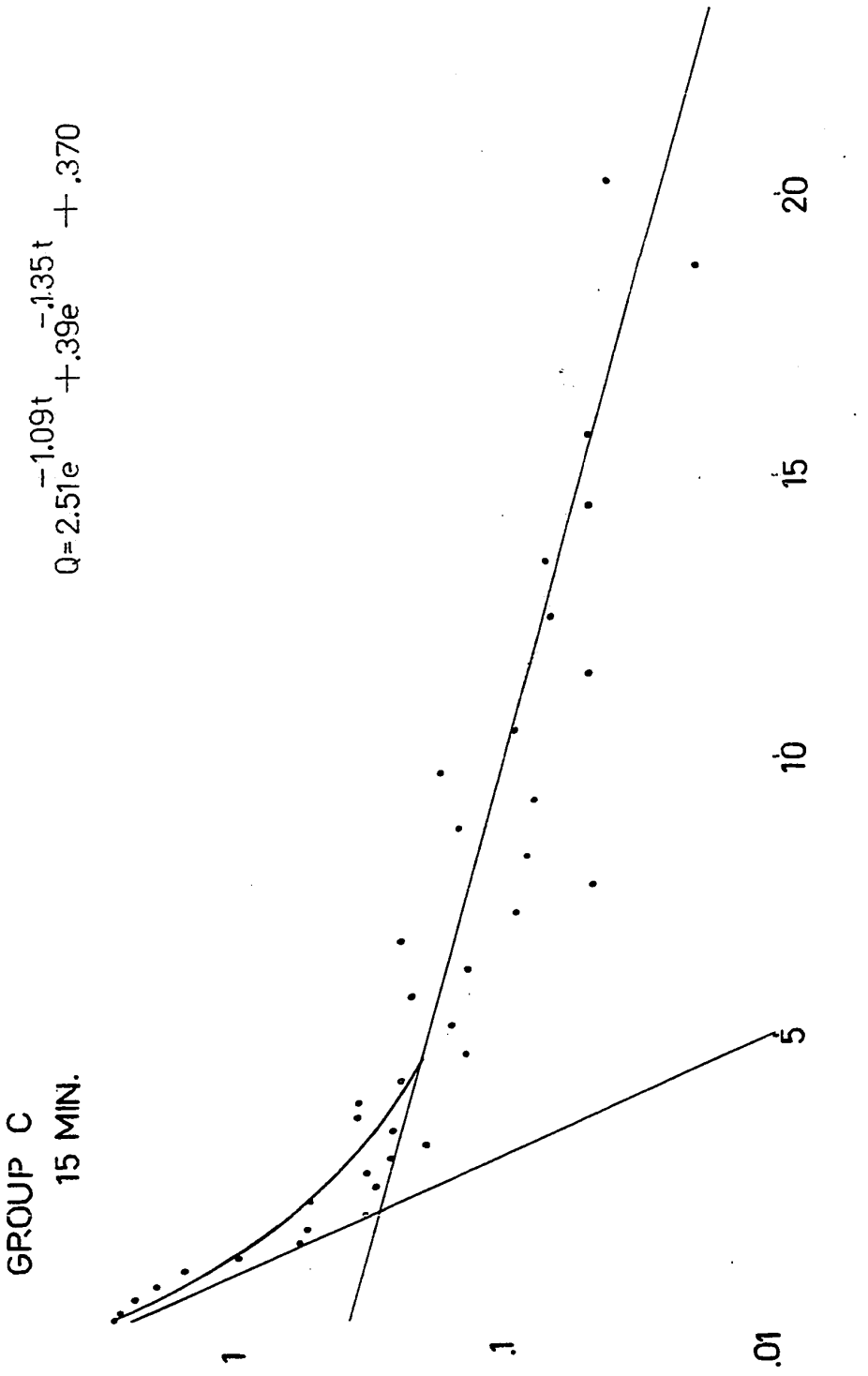
$Q = 1.78e^{-1.60t} + .48e^{-.158t} + .400$

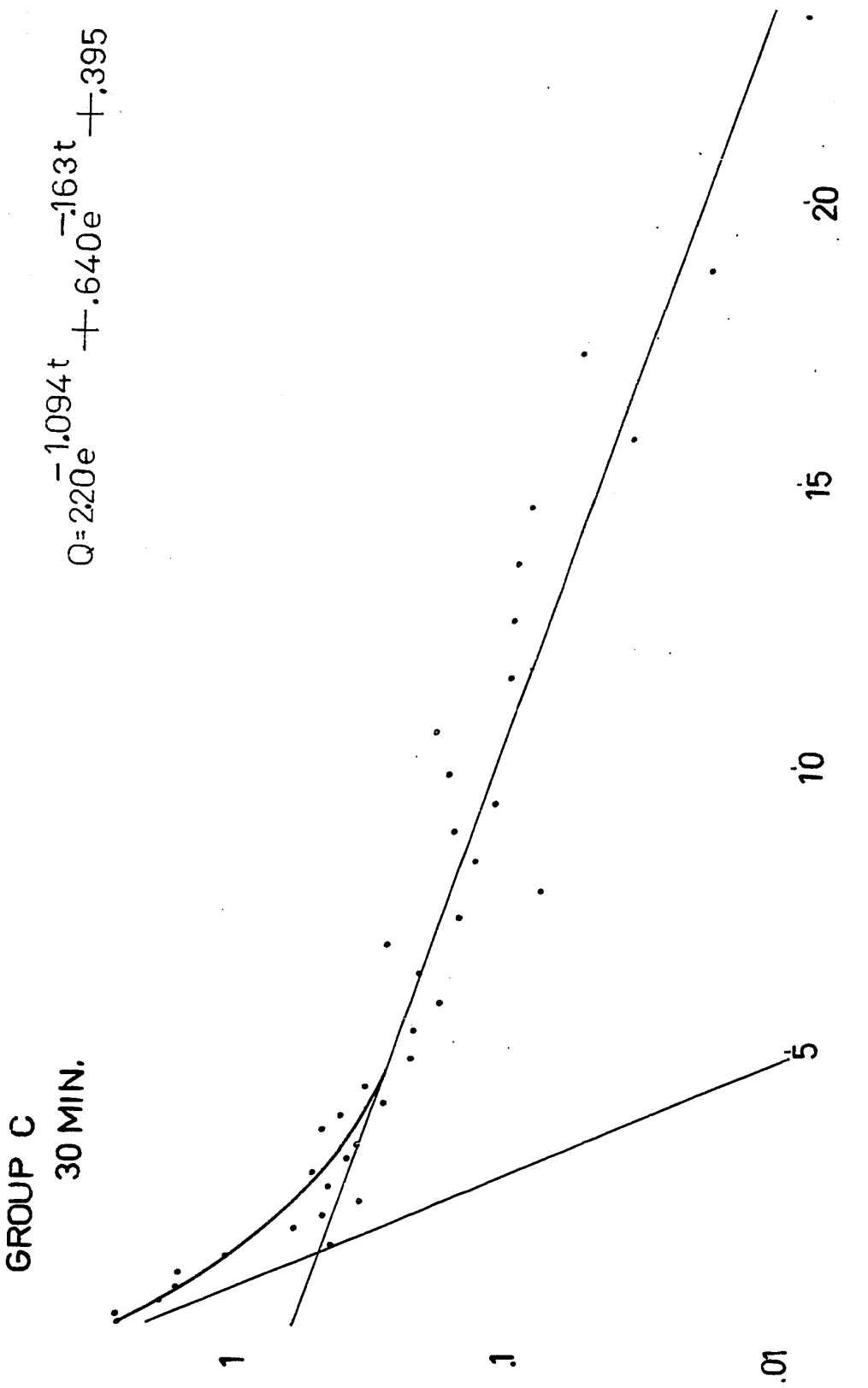



GROUP B
15 MIN.
Q= 1.85e^-1.223t + .30e^-.126t + .400
1
.1
.01
5
10
15
20


GROUP IB
30 MIN.
$Q = 1.67e^{-.945t} + .44e^{-.190t} + .400$
1
.1
.01
5
10
15
20


GROUP C
5 MIN.
Q=2.31e^-1.188 t + .48e^-.188 t +.400
1
.1
.01
5
10
15
20


GROUP C
15 MIN.
$Q=2.51e^{-1.09t}+.39e^{-.135t}+.370$
1
.1
.01
5
10
15
20


GROUP C
30 MIN.
$Q = 2.20e^{-1.094t} + .640e^{-.163t} + .395$
1
.1
.01
5
10
15
20

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